CLINICAL LECTURES

AND

CASES WITH COMMENTARIES

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BY

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PREFACE

Most of the following miscellaneous papers have appeared already in the Journals and in the 'Transactions' of the Clinical Society of London. Some of these I have reproduced with few or no variations from the original form; in other instances I have more or less freely amended, erased, or enlarged the the text without in any degree departing from the general scope and substance of my meaning.

Although I have ventured to lay this book before the profession at large, my main purpose is to leave it as a legacy to the Middlesex Hospital—*in memoriam.*

53, Queen Anne Street,
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LECTURES

LECTURE I

ON A CASE OF ACUTE RHEUMATISM WITH HEAD SYMPTOMS AND HIGH TEMPERATURE

Delivered May 3rd, 1872. Published in 'British Medical Journal,' August 3rd, 1872.

Richard L—, æt. 21, a pale, delicate, nervous-looking lad, was admitted under my care March 16th, 1872. His father is living, but not strong. His mother died of consumption, and his grandfather is said to have had gout. The boy himself has always been in feeble health. About ten years ago he had an attack of rheumatism, and since that date he has suffered from shortness of breath on any unusual exertion. About two years ago he had a severe cough, with slight hæmoptysis. A week before admission he experienced pain, accompanied by redness and swelling in the left foot.

On admission, pulse 108; temperature 101°.* No definite murmur or rub over the præcordia—only a feebleness and muffling of the heart-sounds, along with the peculiar triple rhythm of pericarditis. Pain, redness, and swelling in the hands and ankles. Ordered a mixture containing in each dose fifteen minims of tincture of perchloride of iron every four hours; nitre poultices to the affected joints, and five grains of compound soap pill in the night. From this date until March 26th the pains in the joints were extremely variable, coming and going in the most capricious manner; they were never at any time remarkable for their severity. The chest complications, however, were severe in the highest degree. The signs and symptoms of pericarditis, with large effusion, developed rapidly; there was unquestionable evidence of sharp pleurisy, with extensive pneumonia on the right side, and evidence enough to arouse the suspicion of a limited pneumonia on the left. During this period the maximum of the pulse-rate amounted to 120, that of the respirations to 60, and that of the temperature to 103·3°. There was no albuminuria. During the same period two leeches were applied to the right lower ribs, the seat of the pleuritic pain, and two to the præcordia, which were subsequently blistered.

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* The temperature in this case is always referred to the axilla, where it is not otherwise specified; and the difference between the temperatures in the rectum and in the axilla is always assumed to be Wunderlich's average, seven-tenths of a degree, although it is exceedingly probable that, after removal from the bath, the difference might be much greater. Fortunately this is of no practical moment for the most part in the calculations given.

The diet was liberal under the circumstances, but no stimulants were administered before the 25th, when four ounces of brandy were allowed.

March 26th.—Morning pulse 126; respirations 60; temperature 101·2°. Evening pulse 132; respirations 66; temperature 102°. Face exceedingly pale. Pain in the right side continues; pain in the joints entirely gone. Characteristic rhythm of pericarditis still present. The præcordial dulness reaches the first interspace above; laterally it extends one inch beyond the mammary line at the level of the nipple. Ordered one grain and a half of iodide of potassium in a mixture containing senega, carbonate of ammonia, and squill; two leeches to the painful part of the chest; six ounces of brandy daily; and a sixth of a grain of acetate of morphia to be injected beneath the skin. For diet he was allowed two pints and a half of milk, two eggs, two pints of strong beef tea, a custard pudding, and twelve ounces of bread daily.

27th.—Morning pulse 126; respirations 42; temperature 101·8°. Evening pulse 138; respirations 42; temperature 102°. Pericardial friction audible over the whole of præcordia.

29th.—Pain has returned to the ankles and hands. Evening temperature 103·8°; pulse 138.

30th.—Slept ill. Passed urine frequently during the night. Friction-sounds much subdued. Ordered a draught containing ether and twelve minims of sedative solution of opium. Morning pulse 126; respirations 48; temperature 103·8.° Evening temperature 104·2.°

31st.—Again slept ill. Wandered a good deal during the night, and continued to wander in the morning. Bowels open three times; motions ochre-coloured, semi-solid, acid. A profuse miliary eruption has appeared over the whole body. Area of præcordial dulness slightly diminishing. 1 p.m.—The temperature was now ordered to be taken every two hours. The maximum to day was 103·8°. Twenty minims of hydrochlorate of morphia were given at once, and a pill prescribed consisting of three grains of quinine and half a grain of digitalis, to be given three times daily. 7.30 p. m.—Thirty minims of solution of hydrochlorate of morphia were administered, and at 9 p.m. twenty grains of hydrate of chloral. At midnight chloral was again administered to the amount of fifteen grains, and a draught ordered every six hours containing three grains of quinine and ten minims of tincture of digitalis in lieu of the pill.

April 1st.—Slept at intervals after the night draught without wandering. Maximum temperature 104·2° at 3 a.m.; minimum 102° at 11 a.m. Wanders now from time to time. Ordered a starch and opium enema to control diarrhœa, and an occasional draught composed of aromatic spirit of ammonia, ether, and syrup of tolu to relieve dyspnœa. The quinine and digitalis were repeated at night. Twenty-five grains of chloral were given in divided doses.

2nd.—Maximum temperature 103·8° at 9 p.m., when the pulse marked 132; minimum temperature 102·6° at 10 a.m. Diet on the same liberal scale as before.

3rd.—Slept badly, and wandered much during the night. Subsultus tendinum was noted for the first time this morning. Pericardial friction subsiding. Renewed pains in the hands. Sonoro-sibilant râles heard throughout both lungs, except over the left supra-spinous fossa and in the left axillary region, where there are moist sounds of high pitch. Maximum temperature 103·5° in the evening, when the respirations marked 54 and the pulse 132. Minimum temperature 102·2° at

5 a.m. Six minims of tincture of opium were added to each dose of the quinine and digitalis mixture.

4th.—Wandered during the night, but slept three or four hours. No subsultus. Maximum temperature 103·2° at 9 p.m., when the respirations amounted to 68; minimum temperature 102° at 9 a.m.

5th.—Slightly delirious during the night, but slept fairly at intervals. Again there is subsultus. He picks the bedclothes, and the muscles of the mouth are in constant agitation. Maximum temperature 103·2° at 2 and 9 a.m., and at 10 and 11 p.m.; minimum 102·2° at 6 a.m.

6th, 10 a.m.—Subsultus continues; hands exceedingly tremulous; tongue dry and brown. Quinine increased to six grains—double the former dose—and an ice bag applied to the forehead. This day the temperature throughout maintained an unusually high standard. At 6 a.m. it had reached 104·6°, and although it fell somewhat from time to time, at 4, 5, and 9 p.m. it amounted to 105°. At 10 p.m. it was 105·5° in the axilla, and at 11.23 p.m. 107·2° in the rectum. At the same time the pulse numbered 150 and the respirations 54. The subsultus was now excessive; the delirium and the floccitation continued, and there were extreme pallor and prostration. At 11.25 p.m. the patient was put into a bath.

First bath.—Temperature of bath 90° cooled down to 67°; duration forty minutes. Temperature in rectum at commencement 107.2°, at close 101·8°; minimum temperature after bath 97° in axilla. Amount of after-fall 4·1°. Period of after-fall fifteen minutes. Amount of aggregate fall 9·5°. Period of aggregate fall fifty-five minutes. In the bath the symptoms abated slightly, and there was less pallor. On removal he seemed to be clear-headed and comfortable. He shivered, however, and it was at least half an hour before the shivering was subdued after the application of hot bottles and bags to the trunk and extremities. Ordered brandy and port wine, of each eight ounces. From 12.20 a.m. on April 7th—the date of the minimum degree—the temperature rose gradually, until at 5.30 a.m. it reached 105·1° in the rectum, when he was again put into the bath. At this time it was impossible to count the pulse.

Second bath, April 7th.—Temperature of bath 90° cooled down to 78°; duration twenty-five minutes. Pulse at 5.40 a.m. 150; at close of bath 138. Temperature in rectum at commencement 105·1°, at close 103°. Minimum temperature after bath 99·8° in axilla. Amount of after-fall 2·5°. Period of after-fall twenty-five minutes. Amount of aggregate fall 4·6°. Period of aggregate fall fifty minutes. During the second bath the intellect became much clearer, although the subsultus continued in full force. Twenty minutes after removal the pulse fell to 116, and its characters showed great improvement. At 6.40 a.m. the pulse marked 96; at 6.55 he was quite rational, and conversed with his father about family affairs. At 10.45 a.m. the temperature rose to 103·6°, and at 11 he was ordered ten grains of quinine with ten minims of tincture of digitalis every six hours. At 12.10 p.m. the thermometer stood at 104°, and at 12.30 he was again put into the bath.

Third bath.—Temperature of bath 82°, cooled down to 76°; duration thirty minutes. Temperature in axilla just before commencement 104°. Minimum temperature after bath 98° in axilla. Amount of aggregate fall 6°. Period of aggregate fall fifty minutes.

Fourth bath.—At 8.15 p.m., seven hours and a quarter after removal

from the last bath, he was again immersed, the temperature in the rectum standing at 104·9°, and the pulse marking 144. Temperature of bath 90°, cooled down to 79°; duration twenty-five minutes. At 8.20 the temperature in the rectum rose to 105·4°. At 8.40 he was removed from the bath and rubbed with a warm dry towel. At the time of removal the temperature in the rectum amounted to 102°; in five minutes afterwards it fell to the minimum 98° in the axilla. Amount of after-fall 3·3°. Amount of aggregate fall 6·2°. Period of aggregate fall thirty minutes. On this occasion the temperature rose rapidly from the minimum, and at 1.40 a.m., April 8th, within five hours, reached 104·8° in the rectum. It was thought the rapidity of the rise might be connected with the dry rubbing of the body; hence this proceeding was discontinued ever afterwards.

Fifth bath.—At 1.40 a.m., April 8th, the bath was again administered. Temperature of bath 89°—78°; duration twenty minutes. Temperature in rectum at commencement 104·8°, at close 102·5°. Minimum temperature after bath 99° in axilla. Amount of after-fall 2·8°. Period of after-fall ten minutes. Amount of aggregate fall 5·1°. Period of aggregate fall thirty minutes. He wandered much during the first five minutes, and said he felt cold and chilly. He had an occasional short cough, and at the close a slight fit of shivering. Immediately after removal the extremities were warm, but the pulse could not be counted. Brandy was given freely, and in twenty minutes he was asleep. The temperature rose slowly until 10 a.m., when it stood at 103°. He had slept well for four hours, and now answered questions rationally. The subsultus and the tremors also had abated considerably, but the cough was severe.

Sixth bath.—At 4.15 p.m., April 8th, the temperature reached 104·9° in the rectum, and at 4.35 he was placed in the bath. Temperature of bath 88°—76°; duration twenty minutes. Temperature in rectum at commencement 104·9°, at close 103°. Minimum after bath 97·8° in axilla. Amount of after-fall 4·5°. Period of after-fall ten minutes. Amount of aggregate fall 6·4°. Period of aggregate fall thirty minutes. He complained of cold soon after immersion, shivered, and asked for brandy. Two fluid ounces and a half were given. After removal he looked bright and cheerful. Slept well during the night and rarely wandered.

9th.—At 8 a.m. the thermometer marked 103·6°, and at 10 p.m. 103·8°. By this time the pain and stiffness had returned to the joints. There was no subsultus. Pulse 120, regular, and of much better volume. At 1 p.m. the subsultus reappeared, and there was slight deafness. Ordered eight ounces of brandy and four ounces of port wine daily. 10 p.m.—He had slept much, and was now perfectly rational.

10th.—Passed a fair night, but talked in his sleep. Perspires freely. Seems drowsy, but clear in intellect. Urine abundant. Diarrhœa continues. From 10 a.m. on the 9th the temperature had fluctuated a good deal, on the whole maintaining an average of 102·8°. At 8.35 on the 10th it rose to 104·8°, and at 8.40 he was again immersed.

Seventh Bath.—Temperature of bath 91°—76°; duration twenty minutes. Temperature in rectum at commencement 104·8°, at close 103°. Minimum after bath 97·4° in axilla. Amount of after-fall 4·9°. Period of after-fall twenty minutes. Amount of aggregate fall 6·7°. Period of aggregate fall forty minutes. On immersion he was sensible

to a certain extent. During the bath the tremors abated and at last disappeared. The pulse became steadier and stronger, and, although there still remained some incoherence at the close, the mind on the whole was clearer than before. Fifteen grains of hydrate of chloral were given at 10 p.m. Ordered for diet, a quarter of a chicken, milk one pint, strong beef tea half a pint, and two eggs, daily.

11th.—Slept well after the chloral draught, but wandered in the night. He is rational this morning. Complains of vertigo. Appetite good. No pain or swelling in the joints. Dulness over the lower third of the right back; delicate crackling sounds at the left posterior base. Vocal resonance loud and breathing bronchial over the spine of the right scapula. Skin abraded at the extremity of the sacrum. Still some diarrhœa and subsultus. Maximum temperature 104·4°; pulse 120; respirations 44. 9 p.m.—Perspiring moderately; again delirious. At 9.38 the bath was repeated.

Eighth bath.—Temperature of bath 90°—76°; duration twenty-seven minutes. Temperature in rectum at commencement 103·8°; at close 102°. Minimum after bath 98° in axilla. Amount of after-fall 3·3°. Period of after-fall twenty-five minutes. Amount of aggregate fall 5·1°. Period of aggregate fall fifty-two minutes. During the bath the tremors subsided; after removal the hands and feet were cold but the body was warm. Brandy was freely administered in the bath and afterwards.

12th, 10 a.m.—Slept well without chloral. Pulse 120; respirations 52; temperature 103°. There is much subsultus but less delirium. Marked general hyperæsthesia. Pulse thrilling and dicrotous. Pericardial friction still audible, though faintly. Area of præcordial dulness still more than normally extensive. At 1 p.m. champagne was ordered in place of port.

13th.—Since last note the temperature has never risen to more than 102·6°. Slept well after chloral, but wandered slightly when awake. He is quite sensible now, but drowsy. Complains of pain in the left elbow and knee. Three motions passed involuntarily and unconsciously. Pulse steadier; very slight subsultus.

14th.—Maximum temperature in the last twenty-four hours 103°; minimum 100°. Slept well, but wanders now and seems drowsy. Subsultus continues. One motion, passed naturally. No friction heard over the præcordia. Ordered eight ounces of brandy and a pint of champagne daily.

15th.—Maximum temperature in last twenty-four hours 100·8°; minimum 99·8°. No subsultus, except when he is questioned or disturbed. Mind clear. Takes abundance of nourishment, including oysters and chicken. There is nothing abnormal in the heart sounds.

From the 16th of April the temperature, which during the administration of the baths had been taken every two hours, and since their omission on an average every three hours, was now taken four times a-day and recorded twice only.

18th. — Morning pulse 120; respirations 44; temperature 99·6°. Evening temperature 100·6°. There is considerable hyperæsthesia of the legs, which are very rough and notably wasted. No twitching of the face; slight tremor of the hands. From this date until the 27th of April he remained in much the same condition. On the 22nd the quinine was reduced to six grains every six hours, and the brandy to four ounces daily. At this time the diet consisted of a chop, four

oysters, two eggs, half a pint of strong beef tea, one pint of milk, one pint of pale ale, and twelve ounces of bread daily. On the 26th, in addition to the quinine mixture, a drachm of cod-liver oil and half a drachm of syrup of iodide of iron were ordered to be taken once in the day.

27th.—Evening pulse 114; respirations 36; temperature 99·8°. For the first time since his admission, except after the bath, the evening temperature fell below 100°. It was noted that he ground his teeth during the night. This symptom had been observed by the ward-sister for a fortnight before, but had never been reported. Quinine reduced to four grains every six hours; cod-liver oil and syrup of iodide of iron doubled in quantity and administered twice daily.

May 2nd.—Morning temperature 98·8°. For the first time since admission below 99°, except after the bath. He ground his teeth last night. He had ceased to do so since the 27th ult. Appears to be gaining some flesh.

Sequel of Case.

18th.—Morning pulse 90; respirations 20; temperature 98·2°. Ordered one grain of quinine and five minims of tincture of digitalis twice daily.

19th.—He was wheeled about the ward in a chair for an hour and a half.

20th.—Pulse 78—for the first time below 90 since admission; respirations 18; temperature 98·2°.

21st.—Morning temperature 97·8°

30th.—Citrate of iron and ammonia administered in an effervescing mixture, and continued from this day to the end of the case.

June 17th.—The heart's apex is found beating under the fifth rib, an inch and a half below the nipple and one inch within the nipple-line. First sound muffled and wanting in accentuation; no increase in the area of præcordial dulness; resonance good, and breathing simply weak throughout the lungs.

From May 23rd until the date of discharge the pulse ranged from 76 to 102, and the temperature from 96° to 99·4°. The convalescence was exceedingly slow by reason of the patient's timidity and unwillingness to venture upon the free use of his limbs.

On June 22nd he left the hospital for Eastbourne; fifteen weeks after the invasion of the rheumatism, eleven weeks after the first bath, and ten weeks after the last.

N.B.—Seen by me some months after his discharge in fair health, but with a loud murmur at the heart's apex, propagated to the back of the chest.

Gentlemen,—It has long been a familiar fact to all who are conversant with the history of acute rheumatism, that now and then in a few rare and sparse examples the malady proves fatal by an unexpected outbreak of overpowering nerve-symptoms. You will find ample illustration of this in the works of those who wrote before the days of thermometry. It has been reserved for a later age, and, indeed, for the writers of the

last ten years, to make the discovery that head-symptoms are associated with hyperpyrexia in many rapidly fatal cases of rheumatic fever. There can be no sort of question that the two sets of cases are essentially the same, and only differ in the commentaries given and the hypotheses advanced by the pathologists of past and present epochs. In olden time it was all metastasis, meningitis, and the like processes; now it is all hyperpyrexia or, at all events, an exorbitant stress has been laid upon hyperpyrexia *pathologically*. It is hardly possible to overrate its influence practically and clinically. From a pathological point of view, however, hyperpyrexia is not all in all. Whatever the power of an inordinate body-heat may be as an agent of destruction in these particular cases, any one who examines the records scattered through the journals or collected elsewhere will at once see that the rule is this, the nerve-symptoms, stand first in the order of time, and the hyperpyrexia second. This is the *rule;* I do not say that it is the unvarying *law*. Place hyperpyrexia where you will—at 108°, 107°, or even 106°—and you will find that the rule holds good. Nay, in a large proportion of cases the temperature at the onset of the nerve-symptoms is below 105°, and often considerably below that level; it may be no higher than 102°. For my own part, I am convinced that when the nature of these cases comes to be thoroughly understood this proportion will be vastly enlarged, and when all the finer deviations from the normal behaviour of the nervous system are duly watched and duly recorded it will be found in the end that the approach of hyperpyrexia is hardly ever unannounced by nerve-symptoms. I dare not say *never* unannounced. *Never* is a strong word—not to be lightly said of anything, least of all of a point in pathology.

Clearly, then, on the showing of all experience, the mischief originates in some profound and damaging impression upon the nervous centres, an impression which gives rise at once to the phenomena of cerebral or cerebro-spinal derangement, and to those of exalted body-heat. Those phenomena are in the main collaterally derived from one common source. I am here following in the footsteps of Dr. Herman Weber, but it is no waste of words to go over the same ground again.* The point is one that is much misconceived, both at home and abroad, by many intelligent and well-informed persons, who still cling to the untenable idea that hypepyrexia lies at the root of all the evil. As a matter of fact, the question is settled

* See 'Transactions of Clinical Society,' vol. i.

beyond dispute. There are on record a fair minority of well attested cases wherein cerebral rheumatism has proved fatal without hyperpyrexia.

Another question, akin to the foregoing, but not precisely the same, is open to discussion. Be it so, you will say, that the nervous impression takes the foremost place at the start, but at any rate, when there is hyperpyrexia, it is this element that plays the principal part in the destruction of life. I am not so sure of that, gentlemen.

Given a well-marked premonitory stage, when the hyperpyrexia arrives at last there is no essential change in the character of the symptoms; they are still of the same kind as they were from the beginning, although of course they are more intense now that they are come to a climax and the case is drawing to a close. If they change at all in form, the new phenomena are only the crisis and complement of their predecessors; the change is all in the direction of natural development. Convulsions, it is true, collapse, cyanosis, and, above all, coma, may succeed to delirium or tremor, but there is nothing wonderful in this or any hypothesis, whether you regard the surplus heat as the chief agent in the process of destruction, or as a mere symbol and accompaniment of danger to life. Any one who watched the progress of our own case for many days before the accession of anything like inordinate fever-heat, and took note of the delirium, the tremors, the quivering of the lips, and the picking of the bedclothes, must have felt assured that the boy's life was every moment in jeopardy, whether the temperature should rise to the height of hyperpyrexia or not, as much as the life of any one who presented the same symptoms in enteric fever, typhus, pyæmia, or smallpox. We do not want hyperpyrexia to account for death in these cases, and in my opinion there is no proof that death is materially hastened by it. Yes, but we have a proof, it may be said. The bath and the wet-pack, and the ice-bag, reduce temperature and at the same time restore consciousness, and it may be life itself. It follows, then, you will say, that as so much good is done when you remove hyperpyrexia, so when the result is death the hyperpyrexia is mainly responsible for the evil. The reasoning is inconclusive; it is based upon a flaw and a fallacy in the English language, which, however, it would be out of place to analyse here. What do we see in the bath? The two sets of phenomena, the reduction of the temperature and the abatement of the symptoms, run *pari passu*, side by side, in parallel lines, for aught we know to the

contrary never meeting and never reacting upon each other. One of these parallel lines may peradventure start ahead of the other by an infinitesimally small space of time, but which of the two it is that takes the lead, no human being can tell. Let me make myself clearly understood. I am not discarding all belief in evil influences of hyperpyrexia or asserting that it plays no part here in the destruction of life. We may fairly put the point in the following shape :—There may be many factors at work, all conspiring to compass the death of the patient. If the sum total of these factors be overwhelming in their strength there is an end of the matter, and the patient dies; but let the powers of life and death be more evenly adjusted, then the removal of any single factor—say the excess of temperature—turns the scale in favour of life, or, to use a more homely metaphor, takes off the last ounce that is breaking the camel's back. This is one way of regarding the operation of the bath. There is yet another aspect of the question, and one that commends itself far more strongly to my mind, although it does not of necessity rebut the foregoing view or set it aside altogether. Looking at the order of succession so often shown by the symptoms at the onset, we may well suppose that our so-called refrigerating agents really strike home at the root of the entire evil, and directly undo the first damaging impression upon the nervous system by administering a wholesome counter-shock, which controls alike and at once the hyperpyrexia and all the associated elements of death. On the former of these points of view—that which represents the bath as removing the last ounce from the camel's back—you do good *by* refrigeration, through the medium of refrigeration. On the latter—that which makes the bath strike home at the root of all the mischief—refrigeration is in the main a coincidence and only in part a medium for the accomplishment of good, although it is inestimable value as of an index of good accomplished. This last-mentioned *modus operandi* of the bath I hold to be the predominant one in the cure of cerebral rheumatism. I act, then, in the main as all men act in the mercurial treatment of primary syphilis. Every one seeks to touch the gums, as a matter of course, but no one imagines that this is really instrumental in the cure; it is only a sign that mercury has done all or nearly all that can be safely expected from it. Even so I regard refrigeration chiefly and mainly as a sign that I have gone far enough with my remedies, that they have done their duty when pushed to a certain necessary point, and that now it is high time to stop. In other words, I regard the

action of the bath in cerebral rheumatism as closely analogous to that of the cold douche in hysteria or in mania.

Be that as it may, believe what you please respecting the *modus operandi* of the bath; practically it all comes to the same thing. In all cases you are bound to put down high temperature and to keep it down.

I will now give you a list of the phenomena of nervous origin which mark, as a rule, the earlier stages of cerebral rheumatism and foretoken a possible outburst of hyperpyrexia. You will at once see that you must allow a large latitude of meaning to the term nerve-symptom, and be keenly alive to everything that can be fairly included under it. The following are the phenomena: delirium, restlessness, rebellious to treatment; and it may be disproportionate to pain, subsultus, tremors, giddiness, deafness, vomiting, jactitation, twitching or stiffening of the face and limbs, pawing the air, picking the bedclothes, disorderly respiration, stupor, somnolence, sullenness, prostration, unconsciousness, or semi-consciousness, apathy, strangeness and waywardness of manner, vacancy and listlessness of expression, unnatural anxiety and lowness of spirits, even unnatural gaiety and garrulity. In short, any change whatever in word or deed, in look or demeanour. Let any one of these sound the note of alarm, and the thermometer, how slight soever its value by comparison hitherto, becomes invaluable now. In the ordinary course of rheumatic fever three or four times a day may suffice, but now it becomes your bounden duty to apply it many times in the day, how many times it is not easy to say. I will give you roughly my own ideas on the subject. If there be delirium with or without restlessness, I should recommend the register to be taken every two hours; I should look upon a longer interval as unsafe, with a certain reservation for the period of sleep, not that sleep is usually disturbed by the introduction of the thermometer. Perhaps every hour would be a safer rule to follow. After all, your labour will often be lost. Delirium and restlessness for the most part pass away and leave nothing behind. Nevertheless they are now and then true prophets of evil, and their warnings are never to be disregarded. Under these circumstances I should never allow the temperature to go beyond 105° at the utmost before administering the bath. As for the remaining prodromata on the list, many of these speak for themselves and proclaim their own gravity in language too plain to be mistaken. In their presence I should be more vigilant than ever; I should even

take time by the forelock and plunge the patient into the bath the moment the thermometer marked 103°; or, under the same conditions, I might give him a warm bath before it rose to that level. Under all circumstances it is important to have the decks cleared for action, and the bath and all its belongings in readiness, for the distance is sometimes fearfully short between the first ominous rise on the thermometer and the last culminating point that concurs with the climax.

On the other hand, there are nerve-symptoms which may easily be misunderstood, or may pass unnoticed altogether, unless you are aware of their strong significance. Often the patient, who a few hours ago was suffering from sore distress in body and mind, becomes unaccountably bright and jocular. By *unaccountably* I mean that the sudden departure of pain, although large allowance must be made for it, and although it is perhaps the greatest of all possible pleasures, is nevertheless utterly inadequate to account for the overflowing joyousness displayed on these occasions. It is not delirium, it is more like some strange form of fatuity or hysteria. Some degree of fatuity there may be in the milder and more manageable cases from beginning to end, but there is no exuberance of mirth about it; the intellect appears to be simply below par, and the expression simply dull, inane, and apathetic. Deafness is another omen of evil which may inadvertently be overlooked.* Prostration is another serious symptom which may readily be regarded as a natural incident in the course of an exhausting fever, when in reality it is the percursor of a dangerous paroxysm. In these cases I should adhere to the rule given for delirium, the general rule which ought never to be *relaxed* in any case.

If, in reference to the possibility of overlooking the nervous derangements, and in order to make assurance doubly sure, you require collateral evidences of approaching danger, I will mention a few symptoms which are held to possess a peculiar value. A dry, unperspiring skin, especially after free perspiration, a wide-spread and intensely red miliary rash, a rapid abatement of the pain and swelling in the joints, or their

* In the present day deafness may be hastily ascribed to the use of salicine or the salicylates. Unfortunately we cannot do without these remedies; it would be inexcusable to forego their undoubted advantages because they give rise to many symptoms that closely caricature the exordia of cerebral rheumatism. When these symptoms do supervene the best plan is to act on the worst hypothesis and to ply the thermometer unceasingly. It will seldom fail to make the distinction.

total disappearance, as aforesaid, diarrhœa and a copious flow of pale alkaline or neutral urine have all been observed to accompany or to precede the pronounced paroxysm of hyperpyrexia.

Let us suppose now that you have bathed your patient once. When are you to bathe him a second time, or it may be for twenty or thirty times? For the most part you may begin at a lower level of body-heat, always bearing in mind that you must take both symptoms and temperature into account; the greater the severity of the symptoms, and the more rapid the rise in the temperature, the sooner will you have recourse to the bath. The main drawback to a moderate level of fever-heat is the danger of cooling the patient below the mark. A free and wide margin should always be left for the fall in the bath, which we can control, and for the fall after the bath, which we can neither control with certainty nor foretell with precision. As for the temperature of the bath, the lower it is at the start the shorter the required period of immersion will be, but it must be remembered that it is not every one who can bear to be plunged suddenly into a cold bath, and with weak, delicate people, perhaps it is best to begin with from 95° to 80°, and to cool down as far as 70°, 60°, or even below that point, if it be necessary or feasible. In the ordinary run of cases the proper time for removal from the bath will be defined by a body-heat between the limits of 102° and 99°, but in this matter you will have to be guided by many modifying circumstances, by the average you have established or assumed for the after-fall, by the amelioration of the symptoms if any ensue, and by the general estimate you have formed of the strength and staying power of the patient himself. In all cases he is to be removed if he show signs of exhaustion or cyanosis. Of the warm bath I have no experience, but I see no reason why it should not be safely and easily managed. I have said next to nothing about the wet-pack and the ice-bag; I prefer the bath on every ground. Wine or brandy will be required before, after, and it may be during its administration. For all these details, and for many more, let me refer you to the 'Treatment of Hyperpyrexia,' by Dr. Wilson Fox.

Before dismissing the bath, it is important to observe that the chest complications in our case, severe and extensive as they were, including pneumonia, pleurisy, bronchitis, and pericarditis, underwent no perceptible change for the worse in consequence of its use.

I have now to give you a few words of warning with refer-

ence to rheumatic fever. Always ask whether the patient has slept fairly well before he came under your charge, and send him to sleep if he has not. Prolonged sleeplessness may, indeed, be a suspicious sign; it may mark the invasion stage of a dangerous paroxysm; but, more than this, it may of itself go far to originate one. Always let the temperature be taken many times on the first day of visitation. You cannot tell what forebodings of evil omen may have appeared before that day. Never on any account let the temperature go beyond 106°, or even 105·5°, whether you have noticed nerve-symptoms or not. Such symptoms may have been present and passed away for a while unobserved, as I am confident they have done over and over again. Never under any circumstances make light of a rise in the fever-heat, especially a rapid unexplained rise; it *may* mean mischief, it *may* develop into a nerve-storm.

As for medicinal remedies, opium and chloral are the only soporifics and sedatives of any real value. They may be used freely enough during the premonitory period, or rather during the initial stages of a paroxysm, but if I saw the danger-signal aloft on the thermometer, I should put on the brake at once and stop the opium for the time being. I might resort to it again after the decline of the crisis, if the agitation persisted and chloral failed to remove it. The truth is, we cannot afford to dispense with opium, but it must never be forgotten that there is no sharp undeviating line of separation between the comatose and the delirious class of symptoms, that stupor may take the place of excitement, and that death by coma or by collapse is the natural outcome of the attack. Of quinine and digitalis I know not what to say. In my own case, as the boy happened to be doing well, on the principle of letting well alone, I was unwilling to alter one iota of the treatment, and so he remained for days and weeks under the influence of quinine, whatever that might be. I cannot determine its value, and the same thing is true of the digitalis associated with it.* Alcohol and other stimuli in large amount, with a plentiful allowance of such strong food as the patient may be able to take, are of course essential. Above all, it is imperative to avoid bleeding, blistering, and all lowering measures whatever. There is little or nothing in the post-mortem

* Quinine in full doses I have sometimes fancied to be useful as an auxiliary. I have never given it in heroic doses, but I am assured on all hands that, whatever power it may exercise over the pyrexia of enteric fever, it has no corresponding power over the hyperpyrexia of acute rheumatism.

appearances to warrant our resorting to antiphlogistic operations.

The coarse changes that we see with the naked eye within the cranium are few and insignificant; they bear no proportion to the supreme gravity of the symptoms during life. A slight degree of vascularity may or may not be noted in the membranes and substance of the brain, but for the most part there is no lymph anywhere, no pus, no red softening, no extravasation of blood, no embolism or thrombosis. It is to be hoped that microscopic examination of the brain, the spinal cord, and the sympathetic ganglia, may one day reveal or disprove the existence of any finer lesions.

In conclusion, let me recall, in a few words, the main point and pith of my meaning in this lecture. Before all things, I wish you to stand on your guard and look ahead. I wish you to mark well the first faint shadow that a coming paroxysm may cast before it, and not to wait until you are face to face with the fearful reality. By so doing you will save many a brand from the fire, many a life that might be lost by a few hours' delay. Medicine, we have been told, is only the handmaid and guide of Nature. It may be so, as a rule, but if ever the resources of our art are more than ancillary to Nature they are so in cerebral rheumatism, provided always they are brought into action at once.*

* Compare Lecture II and Cases 1 and 2.

CHART OF TEMPERATURE. – Richard L.

March 23&24. 25&26. 27&28. 29. 30. 31. April 1. 2. 3. 4. 5. 6. 6. 7. 7. 7. 7. 8. 8. 8. 9. 10. 10. 11. 11. 12. 13. 14. 15. 16. 17. 18. 19. 20.

F

109° 108° 107° 106° 105° 104° 103° 102° 101° 100° 99° Normal 98° 97° 96° 95°

※R. ※R. ※R. ※R. ※R. ※R. ※R. ※R. ※R.

— —

Disappearance of pain in joints.

Return of pain – much sweating Considerable flow of urine.

Wandering at night – Diarrhœa Profuse miliary eruption.

Wanders at intervals.

Very little sleep – much wandering – subsultus tendinum.

Subsultus – tongue dry & brown.

Flocciation.

Flocciation. Delirium, subsultus. pallor; prostration

Calm and clear in mind.

Subsultus – sweating.

Noisy and delirious Pulse incalculable.

Rational – less tremor.

No subsultus.

Bright and cheerful.

Slight deafness.

Again subsultus.

Drowsiness & semi-consciousness.

Intellect clearer.

Less subsultus – sweats moderately – again delirious.

Slept well after bath.

General pains.

Occasional wandering.

Fæces passed unconsciously

Delirium – subsultus drowsiness

Slight subsultus

No delirium.

Wolfenden & Bass Lithe to the Queen 236 Holborn London.

The baths are denoted thus ※ – R. indicates temperature in rectum.

LECTURE II

ON TWO CASES OF ACUTE RHEUMATISM WITH HIGH TEMPERATURE

Delivered July 5th, 1872, and published in the 'Medical Times and Gazette,' March 15th, 1873.

Case 1.—George B—, æt. 26, was admitted on June 4th, 1872. He had lived well and enjoyed almost uninterrupted good health up to the invasion of his present attack of rheumatism on May 28th. On admission, he presented the appearance of a thin, rather nervous-looking man, of a sallow complexion; articular pains severe; no increase in area of præcordial dulness; apex-beat rather high; first sound weak and short. In the evening the pulse was full and bounding, and the temperature 104°. From this date to the 7th his condition underwent little change; the perspirations were profuse, the pulse ruled comparatively low, the temperature ranged from 101° to 103°; he suffered from sharp pain in the left infra-mammary region, and the heart-sounds were somewhat muffled, but there was no murmur, no friction sound, and no extension of the præcordial dulness. Evening temperature June 6th, 103°.

7th.—Slept well during the night, but talked in his sleep.

8th.—Ordered five grains of compound soap pill every six hours, and tincture of perchloride of iron every four hours.

From the 8th to the 14th the temperature never exceeded 103·4°, although it was always above 100°; the maximum of the pulse-rate was 102, the minimum 84. The pains in the joints subsided, and on the 14th were limited to the left wrist and hand. The sweating continued throughout, and there was now considerable cough with expectoration. A miliary eruption appeared on the 12th, and on the same day he began to be deaf.

14th.—10 a.m.: temperature 102·8°; pulse 96. Complains of feeling weak and deaf, but attributes deafness to perspiration passing into the ears. 3 p.m.: temperature 103·6. 9 p.m.: temperature 104·5°.

June 15th.—Temperature at 3 a.m., 104°; at 9 a.m., 105°; pulse 112; respirations 52. Patient is extremely restless, sleeps badly, and feels prostrate. The miliary eruption is now most intensely developed, and there is continued profuse sweating. Heart's impulse bounding; sounds muffled; resonance normal over the præcordia. Dulness, with marked bronchophony, bronchial breathing, and high-pitched crackling over lower half of right back. Temperature at 11.45 a.m., 105°; at 1 p.m., 105·4°; at 1.30 p.m., 106°, while the pulse marked 124. Although the face is moist with perspiration, the trunk and limbs feel dry and pungently hot. At 2 p.m., temperature in rectum 106·3°; pulse 120.

At 2.5 p.m., the patient was put into a bath after taking an ounce of brandy. He bore the bath exceedingly well, and seemed to enjoy it. Duration of bath thirty-three minutes; reduction of temperature of water, 90° to 83° = 7°; fall of body-heat in bath, 106·3° (rectum) to 101·8° = 4·5°; fall of body-heat after bath 101·8° (rectum) to 99·8 (axilla) = about 1·3° in ten minutes, making a total reduction of 5·8° in forty-three minutes. The benefit of the bath was striking in every respect. In particular, the deafness left him entirely, and never returned, although he was at once ordered five grains of quinine every six hours. Shortly after the bath he began to perspire freely, and sweating has been a marked symptom throughout.

From this date he has progressed most favorably, the only drawbacks having been an attack of pneumonia on the left side in addition to that already existing on the right, the occurrence of signs more nearly than before resembling those of pericarditis, and on one occasion great depression and irregularity of the pulse.

Case 2,—Emily D—, aged 32, unmarried, was admitted on June 15th, 1872. There is nothing of any moment in the family history. Her own antecedents had been good, with the exception of a winter-cough. For a fortnight she had suffered from rheumatism, and for a week from rheumatic fever.

On admission she is described as a full-sized, somewhat flabby woman, with a dull, lethargic expression of countenance. The articular pains were of moderate severity. There were sonorous râles in the chest, but no abnormal character in the physical signs over the præcordia. Ordered twenty minims of tincture of perchloride of iron every four hours, and five grains of compound soap pill every night.

June 17th.—Pains fugitive and fluctuating; profuse perspiration; heart-sounds muffled, but free from definite murmur, endocardial or exocardial.

20th.—Temperature: 3 a.m., 102·4°; 9 a.m., 103·4°. Pains subsiding; slept well; sweats less, although the skin is distinctly moist. No change in the physical signs. Temperature at 3 p.m., 104·2°; pulse 96. The bowels have acted profusely, owing to a powerful purgative inadvertently administered. Patient feels extremely prostrate; she is very talkative, although as a rule she is quiet and even taciturn; she talks coherently, however. The skin of the trunk is dry, but there is a slight moisture on the forehead. Pains now limited to the right knee. Ordered brandy three ounces. At 12 (midnight) temperature 105·2°. Her manner is certainly strange, although there is no actual wandering. She was at once injected hypodermically with a quarter of a grain of acetate of morphia. She then fell into a low muttering delirious state, soon passing into sleep.

21st.—Temperature at 1 a.m., 104·8°; 2 a.m., 105·3°. About this time she awoke, and became exceedingly restless, dozing, however, at intervals. Temperature at 2.45 a.m., 105·7°; 3·5 a.m., 106° (in mouth). Patient collected, but dull and somnolent; face dusky; skin dry. At 3.10 a.m. she was put into a bath at 90°. Duration of bath twenty minutes; temperature of bath at close, 84°; temperature in mouth at commencement, 106°, at close, 102°; minimum temperature after bath, 101° (axilla) at 5 a.m.; amount of after-fall about 1°; period of after-fall one hour and a half; amount of aggregate fall about 5°; period of aggregate fall one hour and fifty minutes. The patient for a while felt comfort-

able in the bath; but at length she complained of cold and coughed slightly. Soon after removal she declared herself decidedly better. There were, however, loud sonorous râles over both sides of the chest in front, the vibrations being perceptible to the touch. At 3.40 a.m. she was ordered five grains of quinine and ten minims of tincture of digitalis every six hours. At 8 a.m. the temperature had reached 103·8° in axilla; at 8.15, 104·2° in the mouth. The skin was again dry. Ordered cold sponging to the trunk. At 8.30, temperature 103·2° in mouth; at 11, 104·2° in axilla. The cold sponging was then repeated, with much relief to the patient. At 11.20 a.m., temperature 103·8°; at 3 p.m., 104°. A quarter of a grain of acetate of morphia was at once injected, and the daily allowance of brandy increased to five ounces, At 4 p.m., temperature 104·6°; at 5, 104·7°. No sleep since injection. Fifteen grains of hydrate of chloral administered at once, and five ounces of wine ordered daily in addition to the brandy. At 7 p.m., temperature 105·2°. Cold sponging repeated. At 8, temperature 104·6°; at midnight, 103·6°.

22nd.—At 10 a.m., temperature 102·8°. Slept about two hours. Pains in joints more severe and more extensive.

From this time forth the temperature subsided, and although the patient continued to be restless for some days, and to suffer from a harassing cough, the result of pneumonia and bronchitis, she made a good recovery.

Gentlemen,—It is impossible not to recognize in each of the foregoing cases—at all events, in the second case—a strong likeness in miniature to those fearfully fatal attacks of hyperpyrexia with head-symptoms which startle us sometimes in the course of acute rheumatism. The points of resemblance may be summarized in the following epitome of the report:—In George B— there was a pre-existing nervous diathesis, if I may so speak, and that alone is a preliminary of the utmost significance. The first special symptom to attract observation was talking in sleep on the night of June 6th—not much, it may be said, but quite enough to put us on our guard, and accordingly increasing attention was paid to the temperature. From the 8th to the 14th, the pain in the joints subsided, and almost disappeared. On the 12th there was a red miliary rash, which on the 15th became intensely well pronounced. On the 14th the patient complained of weakness and deafness. The latter symptom, which really began on the 12th, he wrongly ascribed to the influx of perspiration into the ears, for the deafness vanished after the bath, in spite of the free exhibition of quinine, while the perspiration continued as profuse as ever. On the 15th there were exteme restlessness, insomnia, and prostration. At midday the sweating abated, and the trunk and limbs became dry and pungently hot to the

touch. At 1.30 p.m. the temperature reached its maximum, 106° in the axilla. At 2 p.m. the bath was administered, and all the threatenings of mischief swiftly passed away. In the case of Emily D—, on June 17th it was noted that the pains were fugitive and fluctuating, and on the 20th that they were decidedly on the decrease. On the same day there was a rise in the temperature, although it was far from amounting to hyperpyrexia; there was rather sharp diarrhœa, probably the result of severe catharsis; there was prostration, possibly the result of the diarrhœa; there was dryness of the skin; and the patient became unusually loquacious, although perfectly sensible in her remarks. At midnight her manner was unequivocally strange; and now the temperature amounted to 105·2° in the axilla. After an injection of morphia she fell into a state of low delirium, with alternations of sleep and restlessness, until 3 a.m. (June 21st), when she became more rational, though dull and somnolent. At this date the temperature, which had been rising rapidly, culminated at 106° in the mouth. Again, you will observe the prodromata, or rather the inaugural symptoms, are in accordance, as far as they go, with those of the *grand mal*; and again, the bath which was promptly administered gave complete relief, at least when supplemented with cold sponging, which cleared away the last remnants of fever and excitement. It might, indeed, be alleged that many of the so-called prodromata in Emily D— were symptoms of poisoning by morphia, and nothing more. This can hardly have been the case in any material degree, for the morphia injection was repeated in the same dose within eleven hours, and produced no corresponding consequences. Even if the morphia be held responsible in any degree for the results in question, it can only have been collaterally and secondarily responsible. The main mischief lay in the nerve-centres and in that loss of nervous equilibrium which gave force and fulness to the operation of the morphia in the first instance, while the succeeding calm and the restoration of the balance after the bath made it powerless for evil in the second. Again, it might be alleged that pneumonia is responsible for the attack in one case, and over-purgation in the other. It may have been so in the beginning, but without rheumatism the symptoms would never have presented the peculiar features recorded; and with rheumatism, if they had been allowed to run their course, there is no reason in the world why, having advanced so far, they should not have gone a step further and ended in hyperpyrexia and death. Clearly, then, insignificant as

the above attacks may appear at first sight to those who have never had the good or the ill luck to come face to face with the worst manifestations of the malady, they really raise a question of the highest importance. Were they nothing more than mere rudimentary disorders, essentially innocuous, and never, under any circumstances, destined to come to a dangerous maturity, or were they grave and formidable attacks in embryo, cut short in their development by medical interference? On the whole, I am convinced that the greater and the lesser seizures are the same in nature; whether they are the same in tendency, what their comparative degrees of danger may precisely be, how many times the lesser evil, if let alone, may merge in the greater—these are questions I dare not pretend to determine. Anyhow, even if we believe in a difference of tendency, it would be unwarrantable to act upon that belief. The commencement of a grave case must strongly resemble the consummation of a mild one; and, difficult—or, rather, impossible—as the distinction must be on the moment, it is the bounden duty of the physician *for all practical purposes* to assume their identity in every respect.* In other words, if in acute rheumatism he meet with a temperature of 105° or even on occasions 104° or 103°, along with nerve-symptoms and other congenial phenomena, he should at once plunge the patient into a bath. Chest complications need not deter him, nor even a considerable amount of prostration. For the average run of cases, the plan recorded in the notes may suffice; it is slow, indeed, in accomplishing the end in view, but it is safe. If ever the old maxim "*Principiis obsta*" is to have any value, it should have it now under the circumstances above given; and if ever delay is to be dangerous, it should be dangerous now. We cannot afford to wait for anything like an outburst of true hyperpyrexia, whatever that may mean, although, of course, it would be a far grander achievement to extinguish than to forestall one, and although, of course, whenever preventive measures succeed, it is easy to say in the end that there was nothing to prevent from the beginning. There is no hard-and-fast definition of hyperpyrexia in the nature of things; what is only pyrexia in one man may be virtually

* Dr. Wilson Fox appears to have found that spontaneous recovery in acute rheumatism after 106° is rare, and *almost* unexampled. If Dr. Fox is right, the question of absolute identity in all respects is well-nigh settled beyond controversy, at least as far as my own cases are concerned. (See Dr. Wilson Fox on the 'Treatment of Hyperæmia,' p. 27, and foot-note.)

hyperpyrexia in another, with all its associated elements of danger to life. Given corresponding symptoms, I regard 105° as the utmost limit allowable before the adoption of the bath, and even without any ascertained history of corresponding symptoms I should never wait beyond 106° or even 105·5°. I have made no reference to medicinal or other measures preliminary to the bath or supplementary to it, partly because it was beyond the scope of the present lecture, partly because no long time ago I entered at large in the particulars of the subject. In conclusion, I venture to express the hope that, with our attention awakened to the possibility of forthcoming mischief, we shall hear less and less of the *grand mal* from year to year; and that, as it appears to be absolutely unknown even now in many parts of the world, so hereafter it shall be comparatively unknown elsewhere.

LECTURE III

ON A CASE OF ACUTE TUBERCULAR DISEASE, WITH OCCLUSION OF THE VAGINA.

Delivered May 24th, 1872. Published in 'Lancet' August 3rd, 1872.

Eliza W—, æt. 15, was admitted under my care on the 26th of April, 1872. Her father and mother are alive and healthy, and there is no history of consumption in the family. The patient herself had always enjoyed good health until about six months ago, when she experienced acute pain in the head, recurring from time to time. For the last three weeks her fellow-servants had noticed that she was stupid and dull; previously she had been an intelligent and bright girl. On the 22nd of April she complained of severe headache, pains in the back and loins, sickness, and constipation. For some indefinite period she had been subject to a slight harsh cough. She had never menstruated. On admission, the tongue was foully coated; complexion purplish; expression dull; no cough; no dyspnœa; anorexia, but no thirst; complains chiefly of headache, nausea, and pains across the loins, in the knees, and under right axilla; a few large pustular spots on the trunk; nothing of any moment discovered on examination of the chest. Ordered an effervescing draught, with spirit of chloroform, every six hours, and a warm bath.—Evening: pulse, 96, respiration 18, temperature 99°.

April 27th.—After a sleepless but not delirious night, the patient seems drowsy this morning, When moved and examined by percussion and auscultation she is fretful, and apparently hyperæsthetic; pupils dilated; no headache; tongue densely coated orange and white. No urine passed since admission. Twenty-five ounces withdrawn by catheter; specific gravity 1015; contains no albumen. Morning pulse 96; temperature 98·9°. Ordered, two grains of calomel at once, and in three hours a draught of castor oil. Twenty grains of bromide and two grains of iodide of potassium added to each dose of the effervescing mixture. Hydrate of chloral, fifteen grains, administered at night.

28th.—Slept well after the chloral, but tosses her limbs about this morning, seems stupid and heavy, and scarcely answers questions at all. Temperature 98·4°; pulse 102, unequal in force and rhythm. Ordered dry cupping to the nape of the neck, and a castor oil enema.

29th.—Lies in a semi-comatose condition. Jactitation continues. Urine, after retention for twenty-four hours, withdrawn by catheter; specific gravity 1025; reaction acid; contains a trace of albumen. Motions after castor oil abundant and extremely offensive. The head to be shaved, and compound camphor liniment, with the addition of

half its volume of strong solution of ammonia, to be applied to the scalp; enemata of strong beef-tea—three fluid ounces with an egg—to be administered every four hours—3.30 p.m.: patient grinds her teeth; complexion intensely livid.—Evening: pulse 138; temperature 99·6°.

30th.—Complete unconsciousness; extreme lividity. Pulse 146; respirations 54; temperature 101·8°. Died at 3 p.m.

Autopsy, twenty-four hours after death.—The following is an abridgment of Dr. Robert King's report:—Body well nourished. Both lungs, as well as their pleural membranes, were thickly studded with gray miliary granulations. There were also numerous subpleural ecchymoses. In both lungs were small patches of extravasation, and a few islets of lobular pneumonia in an early stage. The right lung was gorged with black blood; the left was almost equally congested, but with blood of a brighter red. The under surface of the diaphragm and the capsule of the liver were copiously beset with miliary granulations. The liver itself contained a number of cheesy bile-stained bodies, a little less than a split-pea; these were found to consist of bile-ducts, nearly if not entirely obliterated by deposits within and around their walls. Granulations were found abundantly in the spleen and kidneys, and in both organs they had partially undergone the cheesy metamorphosis. There was no ulceration of the intestines, but specks of tubercle were discovered on some portions of the peritoneal surface, especially on that forming the recto-uterine fold. The uterus and ovaries appeared to be healthy; but around the os uteri was a little patch which seemed to be roughened by miliary granulations. The vagina formed a huge sac, measuring eight inches in circumference, and containing some twenty-five or thirty fluid ounces of dark, grumous, offensive material, which escaped in abundance on cutting through the thick, tough, and imperforate hymen. Gray granulations were met with at the base of the brain, in the fissure of Sylvius, and on the upper surface of the cerebellum. The brain-tissue was extremely soft, and the lateral ventricles were distended with fluid and greatly dilated.

Gentlemen, at the onset we had the greatest difficulty in ascertaining the previous history of the case. The facts had to be gathered piecemeal from various sources. When gathered they were meagre in the extreme, and we were left in great measure to our own resources. There were many features of hysteria about the case, but a moment's reflection sufficed to dispel that idea. The tongue alone, densely and foully coated as it was, gave positive evidence strongly in favour of more serious mischief, while the absence of all quivering of the eyelids bore witness to the non-existence of hysteria. Indeed, I should rate the negative evidence as even stronger than the positive; for in my experience, amidst all the fleeting phases of that wonderful malady, this quivering of the eyelids is almost invariable. We were, then, compelled to look for some other way of accounting for phenomena which, apart from

hysteria, were in the highest degree alarming. With some misgiving and some mental reservation, I wrote down the diagnosis of tubercular meningitis. When, however, I learned that there was a history of severe and recurring headache six months ago, I began to waver in my decision. It seemed natural to connect that headache with the existing disease; and if there were grounds for assuming this connection, it appeared impossible to retain the original diagnosis, for tubercular meningitis would assuredly have done its work and taken the life of the patient long before six months were over. I wavered then for a while, and bethought myself of abscess or tumour in the brain, but I never erased the written words from the card, and the last thirty-six hours of the girl's life proved conclusively to my mind that the old diagnosis was the true one. During the whole period of her residence in the ward she had been remarkable for the purple flush of her complexion. Now, however, she became intensely livid, and even cyanotic. You are aware that death by apnœa and death by coma are formally distinguished from each other, and they are truly distinguishable at their origin, but at their close they present many phenomena in common, and in the dead-house the appearances in the lungs are much the same. It might, therefore, have been argued with some show of reason that the intense lividity of the girl betokened nothing more than coma assuming naturally the characters of apnœa at its close. The argument would have been untenable. The lividity had existed at the onset long before the coma began, and in the end it was intensified in a degree, and for a period of duration, utterly inexplicable on the hypothesis of coma alone. There must inevitably have been all along intrinsic disease of the lungs quite independent of any pathological process going on within the cranium, and such intrinsic disease by exclusion must have been tubercular. I say by exclusion for this reason: if the lungs on examination tell you nothing at all, or nothing of any special significance, and yet, on general grounds, you are assured of the presence of lung disease, then you may be equally sure that the lung disease is tubercular, and under the same circumstances you may reasonably surmise that there is a free distribution of tubercle, not in the lung-tissue only, but elsewhere throughout the several organs which it usually invades. Amongst these organs the membranes of the brain, in particular the pia mater, occupy a pre-eminent place. Tubercular meningitis, then, was about as safe a diagnosis as any fallible man could make. The old headache was, in all probability, connected with the arrest

of the catamenia, of which I shall presently speak, and stood in no direct relation to the existing symptoms.

If you remind me that there was no history of consumption in the family, I reply that such a history, to the best of my belief, has little or nothing to do with the far-spreading development of miliary tubercle. If, again, you tell me there was no acceleration of pulse or breathing, at least before the closing scene, no cough, no dyspnœa, properly so called, no elevation of temperature, I reply that, strange as it may seem, you *may* have none of these things in tubercular meningitis. With regard to cough and dyspnœa, as the mere diffusion of pulmonary tubercle cannot be discovered by physical examination, so it fails to betray itself outwardly by any violent or explosive act; and in truth it is one of the best marked features of tubercular meningitis that the chest symptoms go to sleep, as it were, while the brain is unnaturally awake. With regard to the pulse I have known at least two cases of acute and universal tubercular disease where the pulse never exceeded 100 from first to last, so long as they were under my observation. Similar remarks apply to the respiration in a minor degree. Understand me rightly, however. I am not going to deny that before the accession of brain-symptoms both the pulse and the respiration may have been, and probably were, above the normal average. Finally, with regard to temperature I have been enabled to declare for tubercular meningitis in a difficult case, on the ground of a temperature never exceeding 99°. Again understand me rightly. I am not affirming the absence of well-developed fever-heat as the rule; far from it. I only say that fever-heat may be absent in some cases towards the close, and even during the whole period of medical supervision.

So much for the aspect of the case during life. Let us now pass in review the two most remarkable features in the discoveries of the dead-house: the wide-spread dissemination of tubercle, and the arrest and imprisonment of the catamenia within the sac of the vagina, for sac it may be called with propriety when its orifice was closed by an imperforate hymen, and its wall presented a circumference of eight inches.

Gentlemen, I need not inform you that modern researches have gone far to set aside the old doctrine so long prevalent as to the origination of tubercle, and have established the general, if not universal law, that tubercle, at least in the disseminated form, is not a neoplasm or primary growth, the result of a peculiar dyscrasia, but a secondary deposit, the offspring of an infective process. The aboriginal source of that infection is

said to be for the most part some material which has undergone the caseous degeneration. Analogous deposits in the form of secondary abscesses occur, as you know, in pyæmia. Now, assuming the truth of the aforesaid law, how are we to account for the acute development of tubercle in our case? On careful examination we found nothing whatever to explain it, save only the collection of decomposing catamenial blood in the vagina. Blood of course contains white corpuscles, and white blood-corpuscles are held by many to be chiefly, if not exclusively, the progenitors of pus-globules. Blood, then, in its retrograde metamorphosis, may be supposed to go through a process akin to suppuration, and on this view no one would have been surprised if we had discovered in our case the evidences of pyæmia. Again, there is but one step between suppuration and caseation; and, indeed, the essentials of the change in question may have actually existed in this girl, although, of course, the appropriate form and consistence were wanting; for condensation was impossible with a fresh flow of blood recurring at each catamenial period. On this view there is nothing unnatural or astonishing in the discovery of tubercle, which, in the absence of all other conceivable sources, may, with extreme probability, be ascribed to the decomposing blood-mass in the vagina. Perhaps, however, I have been refining too much and drawing unnecessary distinctions. It may be that sometimes the same foci of infection give rise to pyæmia and tubercle indifferently, the particular product developed varying with the constitution and surroundings of the patient. Nay, more, it is not beyond the limits of a fair presumption to suppose that both processes may occur at the same time in the same individual, and in our own case it might not be going too far to designate as products of something like pyæmia the pustular scabs on the trunk and the ecchymoses beneath the pleura. There is an obvious reason why the two processes should not *often* be found co-existing in the same person; pyæmia will, for the most part, kill before tubercle has time to grow.

I conclude, therefore, in favour of the disintegrating blood as the fountain-head of the mischief, and this conclusion is positively and strikingly confirmed by the multitude of miliary granulations clustered around the os uteri, and scattered throughout the recto-uterine fold of the peritoneum. If I am right, the case is almost unique, for I cannot recollect having heard or read of tubercle originating in the decomposition of blood.

LECTURE IV

ON A CASE OF OTITIS, WITH ABSCESS OF THE CEREBELLUM AND PYÆMIA

Delivered December 6th, 1878. Published in 'Medical Times and Gazette,' May 8th, 1880.

ANNIE C—, æt. 12, was admitted under my care on October 12th, 1878, with the following history, as recorded in the notes:—Her father died of phthisis, and one sister of marasmus. She herself has suffered from pains in the head for many years. For the last six months she has been growing thinner, and for six years she has had a foul-smelling discharge from the right ear. On October 7th she complained of the head; on the 9th the heart beat violently, and from that time forth she felt drowsy and vomited much, independently of the food she had taken.

On admission, she is described as a fairly nourished, dark-haired, red-lipped girl; cheeks flushed; tongue furred; pupils large, equal in diameter, and equally responsive to light; eyes encircled by a deep-brown areola; pulse small and compressible. Vomits incessantly, and complains of almost incessant headache, chiefly over the forehead. No strabismus; no retraction of the abdomen. Temperature 100°; pulse 112; respirations 28.

The case is far too long for detailed description. I can only give a sketch of the sequel in a general way, with one or two allusions to particular dates. Summarily, then, the main points were these:—Recurrent rigors, thirteen in number from admission to death, a period of eighteen days. Continued vomiting—often a simple regurgitation of clear inodorous fluid, neutral to test paper, while the vomited matters at other times were most offensive in odour. Foul exhalations from the breath and from the whole body. Pain, seated first in the frontal region, then attacking the occiput, ultimately restricted to the right side of the neck, and there accompanied by much swelling and tenderness of the parts around. Spectral illusions and visions of strange forms flitting about the foot of the bed; hyperæsthesia of the surface; dizziness; difficulty of swallowing, and involuntary passage of urine and fæces. Harassing cough and severe pain on both sides of the thorax and across the abdomen. Impaired resonance over more than the lower half of the right back—noted on October 25th; dulness over both posterior bases, with fine crepitus on the left side—noted on the 26th of the month. In the last stage, unconsciousness or semi-consciousness, diarrhœa with fetid evacuations, sordes on the gums, insensibility of the pupils, lividity of the lips, and, on October 30th, death amid convulsions.

The important phenomena of the pulse, the respirations, and the temperature, I have held in reserve apart from the rest, in order to dwell upon them a little more at length. The pulse was variable; never below 100, often inordinately rapid, especially towards the close; the highest rate on record amounted to 163. The respirations also were variable. Always above the average standard even from the beginning, in the end, during the last week, they rose to an enormous number, culminating in 76. Often they were paroxysmal, but the paroxysms never bore the periodical characters of the Cheyne-Stokes type, the ascending and descending form, that which is bounded by absolute apnœa at each extremity of the cycle. The temperature, high at first, but not immoderately so, on the 17th began to look ominous. On the 19th it was taken every two hours. The maximum for that day was 105·2°, and the minimum 99·4°, giving a range of 5·8°. For the succeeding days the four highest maxima were 105·8°, 106°, 106·4°, and 106·8°. The four lowest minima were 97·6°, 97·4°, 97°, and 96·2°. The four oscillations of the greatest amplitude—the four longest ranges—were 8°, 8·4°, 9·2°, and 10·2°.

For treatment iodide and bromide of potassium along with occasional opiates were tried in the first instance, but soon superseded by opium in repeated doses—five minims of the liquor opii every three hours, or forty minims in the course of the day. The compound ether and compound cetaceum draughts followed in association with the linctus morphiæ, which henceforth did duty for the opium, inasmuch as the pain was now on the decline. At the last, hydrocyanic acid and bismuth were given to control vomiting. Throughout the case most of the ordinary local remedies were in constant use, but leeches were only once applied.

Autopsy from the report of Dr. Sydney Coupland.—The longitudinal sinus contained a small quantity of non-purulent blood clot. The pia mater on the convexity was smooth and transparent, but its vessels were very full of blood. The outer and upper part of the cerebellum on the right side was found adherent to the dura mater, which at the spot presented a dirty slate-blue appearance. The cerebellum itself was similarly discoloured in the same region, and, on detaching it, a quantity of bright grass-green pus escaped from a rent corresponding to the site of an abscess in the white matter of the right cerebellar lobe. The abscess was about the size of a chestnut, and abutted on the surface, a flattened lamina of grey matter alone intervening. The pia mater at the base of the brain was natural, but very vascular. No other abscesses were met with, but seated in the substance of the left corpus striatum, and projecting into the lateral ventricle, was a small opaque-yellow, firm, encapsuled nodule, believed by Dr. Coupland to be a tubercle, and not an old abscess. In the right posterior fossa of the base of the skull the dura mater covering the petrous bone was thickened, discoloured, and in part separated from the bone by a collection of pus. In the immediate neighbourhood of this change the lateral sinus for the lower half of its course was completely obliterated, or rather converted into a narrower channel filled with pus and surrounded by thick soft walls. On examining the neck there was seen to be considerable swelling and matting together of the tissues on the right side around the track of the jugular vein and beneath the anterior margin of the sterno-mastoid muscle. A large group of reddened and swollen lymphatic glands imbedded in fat covered the vessels

from the mastoid process to a level with the thyroid body. The jugular vein throughout its entire course was bounded by a firmly adherent thrombus of a dusky gray colour, and permeated by a narrow pus-containing channel. The thrombus terminated at the junction of the jugular with the subclavian vein, and from this point onwards along the innominate a non-adherent, partly decolorized coagulum extended into the vena cava and right auricle. The right ventricle also and the left auricle contained coagula of recent origin. The heart's tissue appeared to be healthy.

In the right pleural cavity was found fully a pint of chocolate-coloured foul-smelling fluid. The pleura itself was coated above and on its posterior surface, by a thin ash-gray false membrane, below and anteriorly by a thick layer of soft granular lymph. Beneath this last-named layer of lymph were found the remains of a pyæmic abscess in the lung, and the whole of that organ was studded with pyæmic infarctions in all stages of development, from small firm nodules of hæmorrhagic nature, and other nodules, purulent or puriform in the centre, and dark-red at the circumference, to abscesses of the size of hazel nuts, with ill-developed lining membranes. Between the infarctions and the abscesses the lung-tissue was intensely engorged. Beyond a small area of vascularity there were no appearances of pleurisy on the left side. There were, however, numerous capillary ecchymoses in the visceral pleura, and the left lung was pervaded with pyæmic formations as freely as its fellow. The peritoneum was natural. The spleen was large, pale, and soft. The liver was pale on the surface, and on section presented a swollen, glistening character, the lobules being ill-defined, and the consistency of the organ diminished. The cortex of each kidney was infiltrated and bloodless, contrasting strongly with the pyramids, which were full of blood.

After the autopsy the temporal bone was examined at leisure, and sections made through various parts of the petrous portion. The internal auditory meatus contained a small quantity of pus, and its lining membrane appeared to be thickened and discoloured. The membrana tympani also was thicker than natural, and at its lower margin showed a perforation which would admit a probe. The tympanic cavity, as well as the vestibule, the semicircular canals, and the cochlea, were filled with a whitish-yellow caseous-looking material, evidently cemented pus. The walls of the tympanum were discoloured, and some slight discoloration was seen in the upper portion of the cancellous tissue of the mastoid process, and on the floor of the lateral sinus, where it grooves the temporal bone.

Gentlemen,—In the foregoing record of the post-morten phenomena you see portrayed in their most characteristic features the consequences resulting from caries of the temporal bone, or rather I ought to say from otitis interna, understanding the term broadly as embracing all the structures of the auditory apparatus within the boundary of the membrana tympani. Those features are the following:—Pachymeningitis or inflammation of the dura mater, abscess of the brain,

thrombosis of the intracranial sinuses, and pyæmia. Lepto-meningitis, or meningitis simply so called—*i.e.* inflammation of the pia mater—is not so commonly or conspicuously present as its predecessors on the list, at any rate in the adult. Strictly speaking, there was no lepto-meningitis in our case, only a high degree of vascularity in the membrane.

The following are the principal symptoms ascribed by authors to abscess of the brain :—Pain in the head, often agonizing, convulsions, paralysis, coma, drowsiness, stupor, shivering, pyrexia, delirium, vomiting, involuntary passage of urine and fæces ; in a few cases, defective articulation ; in still fewer, loss of language or aphasia. Be it understood, however, that these are the symptoms of the acute stage; for in many instances—perhaps in the majority—suppuration may be slowly progressing without a trace, or with hardly the trace, of a symptom to betoken its existence. I have many times in this place dwelt upon the marvellous apathy and long-suffering of the brain—the great centre of sensation and voluntary motion—under circumstances which one would suppose sure to awaken its resentment. Morbid products, so long as their growth is slow, may lie imbedded in the brain-substance for many months, unknown and unsuspected, until some determining cause occurs to light up an explosion. You are at liberty, therefore, in a large proportion of cases, to recognize two stages in the career of encephalic abscess—the stage of delitescence, and the stage of activity ; the period of smouldering, and the period of conflagration. The first period may be of indefinite duration ; the second is variable, lasting from a few days to several weeks.

Abscess of the brain, so far as it falls under the province of the physician, or more precisely, so far as it is unconnected with injury to the skull, may be said to occur in two principal varieties, one originating in disease within or around the bones of the cranium, the other in pyæmia. A few cases there may be of idiopathic abscess ; certainly a fair number are on record whose origin was never ascertained. These abscesses, however, were for the most part recorded at a time when the pathology of the brain was ill understood. Apparently the inflammations whence they arose were looked upon as accidents which might befall the brain, exactly as pneumonia or bronchitis might invade the lung from causes unknown or incidental to every one in the common course of life. As our pathology advances, these unexplained cases retire into the background, and almost, I will not say entirely, disappear. Sir William Gull would

seem to dismiss them altogether, and Rindfleisch is equally decided on the point. Cases, it is true, have been clearly traced to ulcerative endocarditis in the septic form, but between this form of endocarditis and pyæmia the line of demarcation is so dim as to be nearly invisible, and the cases themselves offer no valid exception to the rule. For all practical purposes, then, the distinction may remain as it stands. Our own case offers an example of the first-mentioned variety—that which originates in the cranial bones—although it is a case of undoubted pyæmia. Let me enlarge upon this point. The case was one of pyæmia, but the abscess in the brain was not pyæmic; it was the direct consequence of disease within the petrous bone, pachymeningitis, and thrombosis of the lateral sinus; it was developed by contiguity alone; its propagation was purely local, and in no degree systemic. The true secondary products of pyæmia were found in the lungs and in the pleural cavity. They sprang from the petrous bone and its surroundings; they differed in their mode of derivation from that origin.

I have spoken of the cranial bones and their appurtenances. This requires a word of explanation. In many cases the bony tissue is itself extensively diseased, and the disease may reach the internal surface of the bone, and so attack the meninges and the brain-substance bodily. In some cases the bony tissues are not extensively carious—nay, they need not be carious at all—but the contained cavities are the seat of disease. In this second class of cases the abscess is often seated at some distance from the bone within the interior of the brain, and is presumably due to inflammation of a vein, or to coagulation of blood within the channel of a vein, the coagulum invading the vessels of the meninges, and in the end those of the brain-material itself. For all these points in pathology we are largely indebted to Sir William Gull and Mr. Toynbee. In the present case there was no disintegration of bone, only a discoloration.

I have distinguished, medically speaking, two principal forms of encephalic abscess, one deriving its source from the cranial bones and sinuses, the other from pyæmia. You may naturally ask me, Is the distinction drawn absolutely accurate at all times? May not caries and other morbid processes within and around the cranial bones and sinuses as readily give rise constitutionally to the products of pyæmia in the brain-substance as they give rise to secondary changes in other organs, and as readily as foci of disease in other organs deter-

mine the products of pyæmia in the brain? There is no *à priori* presumption against this view. Multiple and even single metastatic abscesses may be developed in the brain when the source of infection is far distant. Is there any reason in the world why they should not be found there when the primary source happens to be near at hand within the walls of the cranium? The channel of communication would be essentially the same in both cases. The morbific matter emanating from a source within the cranium may indeed attack the lung-tissues, the pleura, and possibly the pericardium, inducing pulmonary infarctions and abscesses, with or without empyema or pericarditis. More than this, it may pass onwards, clear the capillaries of the lungs, and lead to the formation of pus or puriform deposits, sometimes in the liver, more frequently in the joints and their surroundings. Is there any reason in the world why it should not attack the brain in like manner, by the same route, and with the same result? Strange to say, in the whole course of my reading and experience, I am not sure that I have ever met with an unequivocal example in point. In other words, given a lesion in the cranial bones along with suppuration in the brain, and apart from all foci of infection elsewhere, I have never been able to assure myself that such suppuration arose from a pervading systemic blood-change. As far as I know, the rule for the vast majority of medical cases is this: the lesion and the suppuration are on the same side of the cranium and its cavity. Were the suppuration owing to pyæmia, the abscesses would be found on both sides, or indifferently on either side. Surely we ought now and then to meet with an abscess on the opposite side? As a matter of fact I have never encountered more than one indisputable case of the kind, and that a most insignificant one in every sense, so that, speaking within the range of my own researches, I should practically disregard this case altogether, and make the rule absolute. Of course the experience of the profession may contravene this canon. Abscesses, more or less numerous, originating under the above conditions may have been discovered, or may yet be discovered, on the side of the brain opposite to the lesion. This would at once prove my main contention to be wrong, and all its corollaries and the distinctions founded upon it would fall to the ground. If, on the other hand, that contention is right, then it is a marvellous fact, and one that passes all understanding, when we reflect upon the close relationship between pyæmia and bone-disease. It is true, abscesses of the brain might be ascribed—nay have

been ascribed—to a local and limited pyæmia, and it must be admitted that they present some analogy on a small scale to the phenomena and processes of general blood-pollution, when they are deep-seated and separated from the offending bone by a broad barrier of normal nerve-substance. Such limitations, however, in the meaning of the term appear to me undesirable. In my opinion, pyæmia ought to be regarded as universal in its scope and as co-extensive with the blood itself.

Perhaps it would only be fair dealing to take note of certain allegations and assumptions that might be made in antagonism to the views here propounded. It might be alleged that there is scant evidence for the development of secondary abscesses *in any of the viscera* beyond the lungs in the cases under discussion; and, indeed, it would appear that few, if any, derivative lesions are ever transmitted from a focus of *disease* within the bones of the cranium to the spleen or to the kidneys. If, then, these organs escape contamination, why should the brain suffer? it may be asked. Where is the mystery of the brain's exemption from the ravages of pyæmia? All this is nothing to the main purpose; it only widens the field for wonder, and the old dilemma reappears under a new form. How comes it to pass in these cases that secondary appearances are so seldom disclosed in the viscera beyond the lungs, in the brain or elsewhere, save occasionally in the liver? Again, it might be assumed that the brain-tissues in the neighbourhood of the primary lesion are by that very vicinity undermined in structure, and rendered prone to dissolution at the touch of pyæmia. Hence it may well be that pyæmia, engendered in the bone and propagated through the medium of the lungs, will not assail indifferently the one or the other side of the brain, but will fasten by choice on the side contiguous to the lesion—the side predisposed for the reception of the virus. Even in true pyæmia, then, from the nature of the case the secondary changes might be expected to occur in the corresponding hemisphere, and it would be wrong to ascribe unilateral abscess there as a matter of course to local propagation alone. Let this argument go for the utmost it is worth: it only establishes a preference for one side, not an exclusive attachment to it; and nothing short of this will avail to explain the phenomena as the results of pyæmia proper. A certain proportion of cases might fairly be claimed for the opposite side. No such proportion exists, as far as I know. One undoubted case of this nature, and one only, have I ever encountered. It was under

the care of Dr. Greenhow. The mastoid cells on the right side were discoloured, and contained pus in the fluid form. Masses of concrete pus filled the right tympanic cavity. The right lateral sinus and jugular vein were occupied by a suppurating clot, the suppuration reaching down to the subclavian vein, where the outlet was closed by a rounded coagulum. Numerous characteristic secondary abscesses were scattered throughout the lungs. A minute abscess, about the size of a grain of wheat, was seated in the white matter of the left posterior lobe, near the inner margin of the convolutions. Now, if this is not to be numbered among the unexplained and inexplicable cases, I am of opinion that it must be traced locally to the disease within the bone. At any rate the entire issue may be formulated thus. It is infinitely more probable that an abscess should for once arrive at the opposite side of the brain by local transmission from the diseased bone, than that it should never more than once arrive there from the same starting point by systemic dissemination. Be that as it may, let Dr. Greenhow's case be a true example of secondary abscess, the outcome of pyæmia from otitis interna, it is but one out of a multitude of cases; add, if you please, my own case, with its small encapsuled nodule, believed by Dr. Coupland to be the remains of a tubercle, they are but two cases among so many, and cannot shake the main distinction drawn between the principal varieties of encephalic abscess—a distinction sufficiently near the truth for the time being, until fresh cases are forthcoming to annul it.

The symptoms usually assigned to inflammation of the dura mater are these:—Severe rigors recurring in paroxysms and often simulating those of ague, high fever, headache, vomiting, intolerance of light and sound, convulsions, and it may be pyæmia. Add to the above paralysis and coma, which are especially apt to follow when the pia mater is involved in the inflammation. No great amount of difference, you will say, symptomatically between pachymeningitis and the acute stage of abscess. Certainly not; but you must take the symptoms as a group, and, given bone-disease, the pathological changes are so often associated that the community of symptoms becomes a matter of no moment in practice. Again, the symptoms of thrombosis will not differ essentially from those of pachymeningitis. You would naturally expect this, inasmuch as the wall of the sinus is only a prolongation of the dura mater, a fold or reflection of that membrane. One point requires to be emphasized. When the sinus is blocked by a

foul decomposing clot, the danger of systemic infection is more imminent than when the channel is unobstructed and free. Our own case is remarkable in this particular, that the coagulum attacked the jugular vein, and in its descent accounted well for the descending pain, which, first assailing the head, passed downwards along the neck, and then occupied exclusively the right side of that region over the path of the thrombus. It was not, however, the thrombus alone nor the accompanying phlebitis that determined the pain in question. The tissues around were all coincidently inflamed and played their parts severally in its production. On the other hand, the disintegrating thrombus played the foremost part in the determination of the pyæmia.

On the pyæmia in our case there is no necessity to comment at length. It belonged to to the unequivocally embolic class. All the coarse and conspicuous lesions were seated on the hither side of the barrier interposed by the capillaries of the lungs. The finer fragments drafted from the decaying clot in suspension or in solution might indeed surmount that barrier and spoil the wholesome crasis of the systemic blood, but the grosser *débris* lodged bodily in the bifurcations of the pulmonary artery, and there established blocks as prone to retrograde and degenerate as the primordial thrombus itself. Corresponding changes ensued in the pleural cavity through simple continuity of tissue.

Pyæmia may be caricatured by a whole host of diseases—in fact, by all diseases which present the phenomena of hectic and typhoid symptoms combined. As a rule, however, the thermometer will set at rest the differential diagnosis. In our case the chart alone was decisive on the point; and when interpreted with the aid of the context, proved, beyond the possibility of a doubt, that the lungs were the seat of dangerous and swiftly advancing secondary processes.

In speaking of the treatment of pyæmia it is unnecessary to dwell on the importance of anodynes, soporifics, stimulants, and restoratives, powerless though they may be for any permanent good. Among real remedies, if they have any existence at all, the most popular is quinine in large—I might say in enormous—doses. Quinine, you are told, is antiseptic, antiphlogistic, antiperiodic, and antipyretic. Now, with all these associated antagonisms it ought to be a very powerful agent indeed. If it have any influence in checking the course of pyæmia it is chiefly in virtue of its antiseptic and antiphlogistic powers. Quinine destroys the life or paralyses

the movements of bacteria, vibriones, and all the legions of living molecules that are believed to infest the blood in pyæmia and its congeners: hence it is antiseptic. Quinine destroys or deadens the movements and migrations of the white corpuscles of the blood, and so modifies the processes of inflammation and suppuration: hence it is antiphlogistic. In one or other of these ways, or in both ways at once, it may save the vital viscera from the taint of corruption, and thus avert the approach of death for awhile, or even, it is said, sometimes for a permanency; but once let those viscera be, "touched corruptibly," and then death is imminent, and the drug is simply thrown away upon the disease. With the antiperiodic powers of quinine we have no concern: there is no such thing as periodicity in pyæmia; quite the reverse. As a pure antipyretic, as a mere refrigerant, apart from all other modes of operation, it is inconceivable that quinine can be of any material use where the blood is so profoundly altered as it is in pyæmia. I repeat, it is mainly useful as an antiphlogistic and antiseptic. Other antiseptics have been brought to bear upon the disease—sulphocarbolates, hyposulphites, and the like; they are all, I fear, unavailing. Recollect, however, that we were not concerned with pyæmia alone, but with cranial and intracranial changes, which called for a different course of procedure, and forbade the unlimited use of the routine remedies. I never gave a single grain of quinine, believing it to be an utterly forlorn hope as against pronounced pyæmia, and fearing that it would only aggravate the vomiting, which I endeavoured to hold in check to the best of my power, but for the most part in vain. I endeavoured also to mitigate pain and to control cough and dyspnœa. These ends we may have achieved in some measure; we may even have prolonged life and postponed death for a few days or for a few hours; but the main purpose of all medication lay far beyond the range of all our resources.

One point deserves to be especially noted. You may be startled, perchance, at the free use of opium in the case of a child, and above all in a head-case, so prone as all head-cases are to terminate in coma. Bear in mind, however, that the child was suffering sheer agony. Bear in mind, moreover, that when a medicine has many properties and powers, and when the requirements of the system demand that one of these shall be concentrated upon a symptom of surpassing gravity, all the remaining powers may lie dormant. To put it in a simpler form: when a medicine, from the nature of the case,

tells strongly in one direction, it may easily fall short of its average mark in another. Opium, for example, is called upon to combat imperious, insufferable pain; it may spend all its force and exhaust itself in the effort; it may have no energy left to narcotize or to endanger life in any other way.

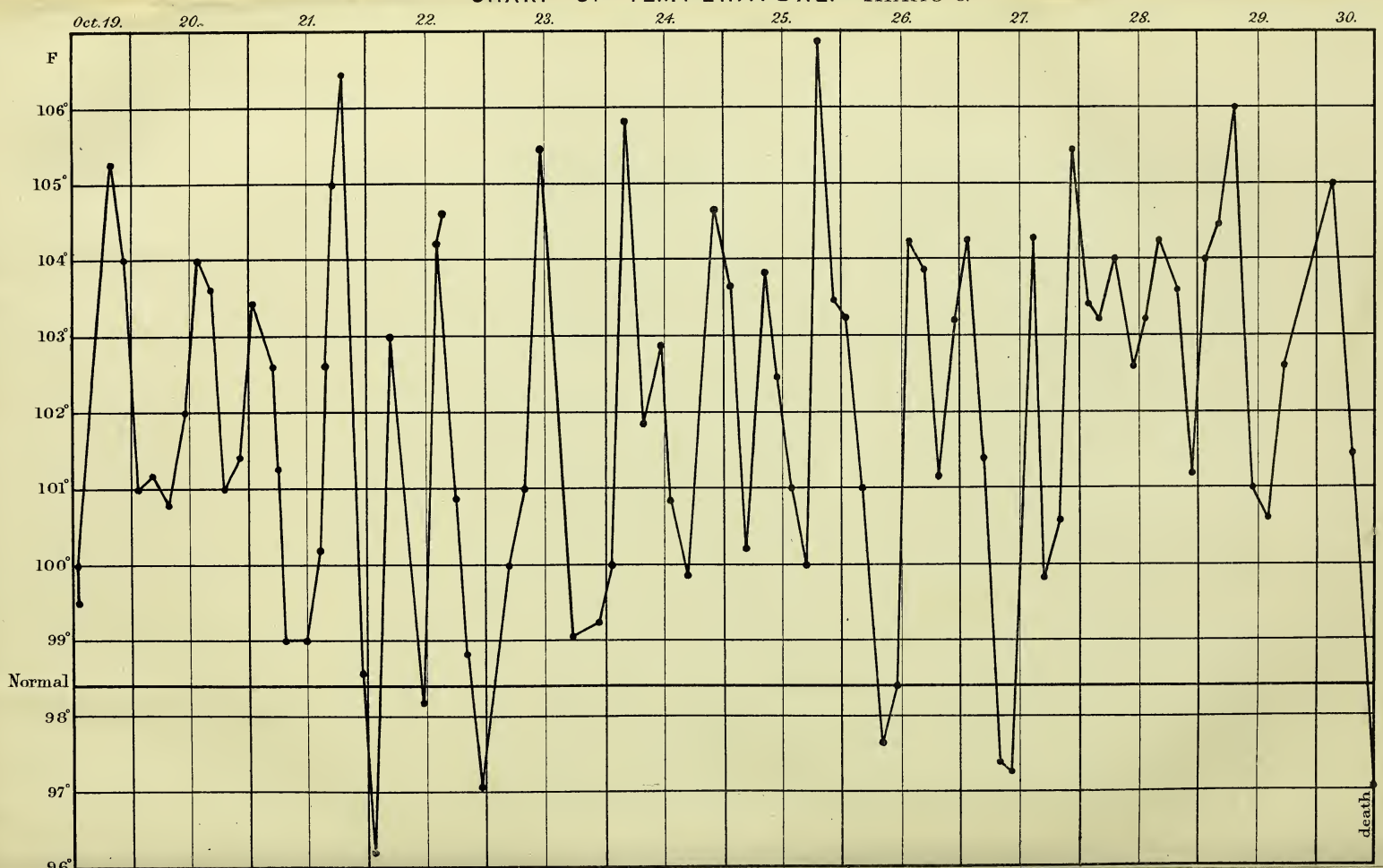
CHART OF TEMPERATURE. – Annie C.
Oct. 19.
20.
21.
22.
23.
24.
25.
26.
27.
28.
29.
30.
F
106°
105°
104°
103°
102°
101°
100°
99°
Normal
98°
97°
96°
death

LECTURE V

ON A CASE OF PERFORATION OF THE TRACHEA BY AN ENLARGED AND CASEOUS GLAND.

Delivered January 16th, 1874. Published in 'Medical Times and Gazette,' January 24th, 1874.

F. S. G—, æt. 4, a frail-looking but most interesting and intelligent boy, was admitted for the first time September 29th, 1873. His father and mother are both delicate; the father has enlarged glands in the neck. There is no history of phthisis in the family. Patient was a first-born child, fat and well-liking after birth; but in a month he began to wheeze, and it used to be said that he was asthmatical, like his grandfather. In November, 1872, he had a low fever, from which he recovered in seven weeks. Shortly afterwards he became an out-patient at Brompton Hospital, under Dr. Powell, and was treated with steel and cod-liver oil. He then went into Oxfordshire, where he caught cold after being out in the damp marshes. At night his breathing became very bad, and his friends thought he would die. On this account he was taken home, and now (August, 1873) the dyspnœa grew worse. He continued to suffer from repeated attacks of shortness of breath, and came at last under the care of the late Dr. Murray, who thought he had an intrinsic laryngeal growth, and on this account admitted him into the hospital under his own care.

On the night of admission (September 29th, 1873) he was seized with severe dyspnœa, stridor, and cough of hoarse, ringing quality, lasting ten minutes, and terminating with expectoration of mucus. On October 9th, the cough was distressing, and he had an abortive attack of lividity and breathlessness. On October 23rd, 24th, and 25th, the breath was exceedingly short, but he derived great relief from the application of hot sponges to the throat. In all these seizures the head was thrown forcibly backwards. On October 30th, when he was transferred to my charge, the paroxysmal attacks were much mitigated, but there were abundant sonoro-sibilant and moist râles in the chest. The hot sponge to the neck, cold affusion to the face, and a draught containing ether and ammonia, generally availed to remove or assuage the dyspnœa. During the early November he improved immensely, and was discharged on the 18th of that month. No satisfactory examination with the laryngoscope could ever be made; he required chloroform for the purpose, and chloroform he could not bear.

He was readmitted under my care, November 27th, 1873, with a return of the bronchitis, but he did not appear to be suffering from much laryngeal discomfort. The old paroxysms of severe dyspnœa were

gone, but still there were a few remaining chest râles, and still he held his head back as before in some degree. The glands in the neck were observed to be swollen, and in particular one large gland stood prominently forward in front of the left sterno-mastoid muscle. For a full fortnight he continued to improve, seldom suffering from any exacerbation of his ordinary dyspnœa. On December 13th, however, the paroxysms began to reappear with some severity, and I added five grains of bromide of potassium to the ether draught, with the view of controlling spasm. On the nights of December 13th and 14th, he was obliged to sit upright in bed with his head thrown back to recover his breath; the lips were blue, and the respirations short. These attacks passed off after the exhibition of the bromide and ether mixture and application of hot flannels. On the 15th, he was ordered liniment of belladonna to the nape of the neck and between the shoulders; the attacks became fewer and milder, but still he appeared to be in great danger. On the 17th, Mr. Morris was summoned, and all appliances were held in readiness for tracheotomy if required. At 5.30 on the morning of the 18th occurred a violent paroxysm, accompanied by lividity, hoarseness, and orthopnœa. Mr. Morris was again summoned, and soon after 8 a.m. performed the operation of tracheotomy, or, more exactly, that of laryngo-tracheotomy. (I quote from Mr. Morris's note). "A large vein taking a transverse course was divided, causing severe hæmorrhage for a short time until the ends were secured. The presence of the tracheal tube greatly aggravated the spasms, which were attended with short expiratory efforts and cough, with twenty long inspirations and several shorter ones between the acts of coughing in the course of a minute. The tube, however, was at last introduced and kept *in situ*, and at 10 a.m. the boy was breathing through it comfortably. At 11 a.m. it had to be removed again. At 3.30 p.m. the respirations were easier than they had been for the last three days—they were fifty-two in the minute; pulse 140; expectoration slight and free from blood-stains. Nothing could be seen by condensed light thrown into the opening in the trachea. Any attempt made to keep the orifice patent by means of curved wire caused dyspnœa and distress." Carbonate of ammonia (a grain and a half) and spirit of chloroform (five minims) were ordered every three hours.

On December 19th, at 9 a.m., the child was sleeping quietly. At 10 the breathing became more laboured, and three ounces of brandy in the day were prescribed. In the evening he appeared to be more comfortable, and towards midnight went to sleep; the pulse marking 148, and the respirations 44.

On December 20th he looked more comfortable still; sat up and played with his toys. There were sonoro-sibilant and moist râles over the upper regions of the chest; below, at the posterior bases, dulness on percussion, with tubular breathing on the left side. The wound was open and discharged mucus in abundance.

From this date to the 24th he continued to gain ground; the pulse, respirations, and temperature all declined; the percussion dulness disappeared at the bases and along with it the tubular breathing in the same regions, although above over the scapulæ there still remained a well-pronounced bronchial and blowing character in the breath-sounds. The wound appeared to be healthy, and, save that the child was unable to breathe freely through the natural passages when the aperture was closed, everything seemed to offer the fairest promises of a successful

issue, for the time being at least, when on the evening of the 24th he became drowsy, and the lips looked ominously livid. He rallied, however, on the two following days; but on the morning of the 27th he lost his appetite, grew livid again, and seemed languid and distressed in his breathing. During the visit he was seized with a severe paroxysm of dyspnœa, accompanied by a sharp cry. Ordered inhalations of nitrite of amyl. At 3 p.m., after partial recovery from this seizure, he was again attacked with another and yet more violent outbreak of dyspnœa, characterized by gasping inspiratory efforts. He died within five minutes.

Autopsy (from Mr. Sidney Coupland's report).—No examination of the brain was allowed. There were some old adhesions between the upper surface of the liver and the diaphragm. On raising the sternum, a collection of enlarged and somewhat indurated glands existed in the anterior mediastinum, mostly over the root of the right lung—one of these glands breaking down into a cheesy mass. In the posterior mediastinum there is a chain of enlarged glands from the size of a pea to that of a hazel-nut, and just above the right bronchus these glands have formed a mass of perfectly caseous matter, while the glands higher in the neck are also slightly enlarged. On laying open the trachea, the tube was found to be occluded just above the point of bifurcation by a mass of cheesy matter extending into the right bronchus, and proceeding from the largest mass in the mediastinum, which had ulcerated through the trachea at this point, the aperture measuring nearly half an inch along the axis of the tube, while for more than half an inch above the calibre of the channel was narrowed by the pressure of the gland from without. In the right lung at the apex on its inner aspect is a cavity with thick, fibrous, reticulated walls, containing cheesy matter. This portion of the lung is adherent to the cheesy mass in the mediastinum. The bronchi are slightly dilated in the upper lobe, and much thickened. The rest of the lung-tissue is more or less solidified, especially in the neighbourhood of the cavity, where it is perfectly and uniformly solid, gray, and smooth on section. The margin of the lower lobe is collapsed and somewhat congested, while the surrounding parts are emphysematous; but the tissue is otherwise healthy. At the apex of the left lung there is a mass the size of a bean, translucent on section, with a few fine opaque granulations, and containing in the centre a small caseous nodule. The rest of the lung is congested posteriorly, pale and anæmic anteriorly, with here and there a few patches of collapse. The mucous membrane of the larynx is healthy, except around the margin of the wound, where it is slightly inflamed. The incision has divided a small portion of the thyroid cartilage, the whole of the cricoid, and the first tracheal ring.

Gentlemen,—In commenting upon the case I have just detailed to you, it is not my purpose to encroach on the domain of the lecturer in medicine, or to enter on the wide field of laryngeal pathology at large. Let it be understood *in limine* that, although I may refer as occasion demands to books and papers on diphtheria, croup, ulceration, and the like processes incidental to the larynx, I am not directly concerned

with any one of these processes, but with laryngeal spasm alone without intrinsic laryngeal disease. You must remember in reference to this point that enlarged glands were discovered in front of the sterno-mastoid muscle; that there was no pain or tenderness over the thyroid or cricoid cartilage; that the attacks were in the main spasmodic, as evidenced by their own peculiarities, and by the remedies that removed them; that the voice, respiration, and cough, though coarse and clanging in the earlier paroxysms, presented no corresponding characters of any moment during the long period of comparative repose that ensued: that whenever the boy took a walk about the ward, he uniformly and persistently held his head backwards; and that in the sitting or recumbent posture, whether simply out of breath or suffering from prolonged and distressing dyspnœa, he invariably maintained the backward inclination of the head until the agony or the discomfort passed away. From all these circumstances combined, I found it impossible to resist the conclusion that there could be no intra-laryngeal disease at all, and that enlarged glands, in the neighbourhood of the larynx and upper air-passages in general, lay at the root of the malady; and this conclusion was exactly confirmed by the post-mortem examination. You see then, that our case stands on a different footing from cases of œdema of the glottis, croup, diphtheria, and all the varieties of laryngitis. There the irritability originates from within the larynx: here it was conveyed from without—*either* from a distance, by propagation along the pneumogastric nerve, or along the trachea itself from the stricture at its bifurcation, *or* from the immediate surroundings of the larynx by pressure exerted on its exterior at the spot. If you adopt the hypothesis of nervous transmission, the present case in many respects bears a close analogy to the disease designated laryngismus stridulus, or child-crowing,—a disease ascribed by one of the earliest writers on the subject exclusively to glandular enlargements, but now well known to arise from a multitude of causes. If you refer the spasm to pressure upon the bifurcation, then you must regard the muscular erethism of the whole tube as simply propagated by continuity of tissue from one point in a given region to another. Lastly, immediate pressure, possibly exerted on the larynx, it is superfluous to descant upon. Take whatever view may seem reasonable to you, it is immaterial to the scope and purpose of the present lecture. Now, assuming the correctness of the above diagnosis, we had no difficulty in explaining the whole series of pheno-

mena, with all their exacerbations and remissions, however strange it may appear at first sight that consequences so fleeting and capricious in their onset and in their departure should all be derived from one permanent cause. So long as the glandular structures remained quiescent and unaltered in volume—so long as there was no unnatural excitability either in the muscular system of the larynx or in the nerve-fibres and nerve-centres which govern the movements of that system,—so long the boy breathed with comparative calmness and ease. When, on the other hand, the glands increased in bulk from transitory hyperæmia, or when anything occurred—as many things may occur in a child—to upset the equilibrium of nerve or muscle, then came the spasm and the paroxysm of laryngeal suffocation. Many, indeed, of the minor attacks *may* have been entirely owing to the impaction of mucus at the seat of the stricture over the confluence of the bronchi, and may *possibly* have involved no element of spasm whatever. Even in some of the stronger paroxysms the same cause may have reinforced the influences of pressure, and lent its aid in the production and maintenance of spasm. Such conclusions or conjectures we are at liberty to base upon the disclosures of the dead-house, but at the bed-side of the patient spasm appeared, if not to represent the sum and substance of the morbid process, at least to constitute the chief danger to life; and these views were amply verified by the whole tenor of the case and by the results of the operation.*

Convinced of the accuracy of the diagnosis, while I endeavoured to control the paroxysmal seizures by means of ether, ammonia, bromide of potassium, and cold affusion, I gave iodide of iron in order to promote the resolution of the glands, or at least to check their ulterior development. At the same time, I was most unwilling to entertain the idea of tracheotomy, preferring to hold the operation in reserve until some imperious necessity should arise to enforce it. Such I believe to be the duty incumbent on the physician, when the fountain-head of the malady lies deep-seated, below the

* Possibly some of the later symptoms may have been due to spasm of the trachea. Dr. Morell Mackenzie informs me that in cases of tracheal stenosis, he has always met with symptoms of spasm, but how far they were owing to spasm of the adductors of the vocal cords, and how far to spasmodic contraction of the trachea itself, he has never been able to determine. In spasm of the trachea the value of the tracheotomy would, of course, vary with the situation of the spasm. In any case, however, in the midst of so much uncertainty, we are bound to take our chance and to operate *in extremis*.

level of the larynx, or, in general terms, whenever it is extra-laryngeal. He cannot hope to cure the disease by opening the trachea—he can hardly hope to afford a respite wherein the disease shall undergo a cure spontaneously or otherwise; for the most part it is absolutely incurable. Why should he hasten to take a leap in the dark? Recollect that tracheotomy is by no means an innocuous operation; on the contrary, it is fraught with many dangers peculiar to itself. On this point I refer you to the works of Trousseau, Jenner, Holmes, Greenhow, Hillier, and others; they deal with the subject at large. I only quote those particular sources of danger which fall within my own range, as before circumscribed. *First* on the list stands ulceration of the trachea from the presence of an ill-constructed canula in the wound. This, however, ought never to occur with the improved appliances devised by modern surgery. *Secondly*, suppurative inflammation may take its point of departure from the lips of the wound, and descend into the cellular tissue of the mediastinum. *Thirdly*, granulations may grow exuberantly around and above the canula, and, if the canula be long retained *in sitû*, may lead to stenosis and even obliteration of the larynx. *Fourthly*, the nice mechanism of the laryngeal muscles, which regulate the admission or exclusion of air, may at length be deranged or destroyed, or the muscles themselves may waste from long disuse, and the paroxysms may return when the canula is removed, if ever the time arrive for its ultimate removal. *Fifthly*, mucus may accumulate in the smaller ramifications of the bronchial tubes, and collapse of the lung may ensue, with broncho-pneumonia and lobular consolidation; all owing to many co-operating causes, but primarily, I believe, and perhaps mainly, to insufficiency in the act of coughing, and that again owing to the presence of an opening in the trachea, which cannot of course execute the natural movements of the glottis. You may try to supplement these shortcomings, and to induce an artificial cough, by placing your finger on the aperture after a full inspiration just at the time when the glottis should close naturally and the chest-walls forcibly compress the air, and then by removing the finger just at the time when to the best of your judgment the glottis should reopen naturally, and the air discharge itself with an explosion. This artificial cough may be useful indeed, and even indispensable, but of necessity it falls far short of the perfection of nature with all her fine adjustments and unerring co-ordinations. Moreover, nurses and attendants on the sick are only human beings, liable to

forgetfulness and to fatigue, and in a long, wearisome, and dangerous case, they can hardly be expected for ever to be taking the right course at the right moment. I have done now with the enumeration of the particular risks incurred. Let me in the last place, allude to a circumstance strongly suggestive of danger in a general way. Tracheotomy, in children at least, has been a sad failure in a large proportion of cases occurring in England, and its results contrast most unfavourably with those reported from the Continent, and even from Scotland. What is the meaning of this anomaly? Is it owing to the possible fact that in France and elsewhere, tracheotomy is more freely and unhesitatingly carried into execution than in England, and that in consequence, while some of the survivors may really have been rescued from death by the operation, many more have simply recovered because they were not killed by it and would have done perfectly well without it? Are we in England less careful than other people in the after-management of our cases? Is it our ungenial climate that is responsible for all our misadventures? Is it possible to imagine that the English thorax and the English larynx and trachea are more feebly organized and more vulnerable than those of other nations? Can it be tamely acknowledged that English surgeons and English physicians are below the standard of their brethren abroad? Surely it would be foul heresy and treason to say this, at any rate; and as for the preceding surmises, whatever element of truth they may contain, to my mind they fail to clear away the whole of the mystery. Unexplained, and apparently inexplicable, it hangs like a cloud over all our deliberations, darkening our prospects and bewildering us at every step.

Now, in the face of all these perils is there not enough to make a man pause before he lays open the trachea when the paroxysms are in the main spasmodic and the lesions entirely extra-laryngeal? There is enough, indeed, to make him pause, but not enough to make him withhold his hand altogether in an emergency. Let us try to elucidate the point by a retrospect of our own case. After the operation it is recorded that, with the exception of one solitary experiment, every endeavour to introduce the canula only provoked intense outbreaks of suffocative dyspnœa; and the same consequences ensued on the insertion of the curved wires. Again, the boy resented strongly the application of the finger to the wound, and even the gentlest pressure made with a fold of lint on each side of the neck at a distance from the spot, with the view of closing

the wound without touching it, appeared to embarrass the breathing beyond his power of endurance. Now, if this acute sensibility of the larynx and trachea, or anything like it, already existed, as there is every reason to believe, before the operation, it is impossible that the boy could have continued without surgical interference to expel the copious muco-purulent masses that now escaped through the orifice, but without the operation must have traversed the ordinary passages, and aroused *in transitu* an amount of irritation which, sooner or later, would have ended in asphyxia and death. In this connection, bear in mind that cough and accumulation of mucus were prominent features in the case from the beginning, and assuredly not the mere consequences of the operation. The conclusion is irresistible; the boy must have died, as everyone believed who saw him in his agony.

Now mark the issue. As soon as the operation was over, and the larynx ceased to play its old part in the work of respiration, then the agony also disappeared. The boy lived and enjoyed life for nine full days, until he succumbed at last under an attack of pure suffocation, unattended or at least uninfluenced by spasm, and solely determined by the outpouring of caseous matter, and the complete occlusion of a part of the trachea already narrowed to one half its normal capacity. But for the perforation of the trachea, he might have lived many days, perhaps many months longer. Nay, more, we may venture to affirm that, had the trachea remained imperforate, and had the offending gland at the bifurcation undergone ulterior changes and diminished in volume, or become encapsuled or imprisoned in a solid framework, like the corresponding masses discovered in the lung-tissue, the boy's life might have been saved permanently. But let that pass; all hypothesis apart, the results actually achieved alone justify, and more than justify, the performance of the operation.

LECTURE VI

ON A CASE OF PNEUMOTHORAX

Delivered June 2nd, 1871. Published in 'Lancet,' September 16th, 1871.

JOHN B—N, a french-polisher, aged fifty-two, admitted May 3rd, 1871, was a very healthy man until three years ago, when he had "jaundice and dropsy." His illness lasted about six months. Ever afterwards he suffered from more or less persistent cough, always aggravated in the winter season.

State on admission.—Pulse 120; respirations 40; temperature in axilla 101·5°. Considerable lividity of face and lips; orthopnœa; expression anxious; slight incurvation of nails, and slight clubbing of finger-ends. Sputa loose. Loud rhonchus, with sibilus and moist sounds on both sides of chest, all most intense and most abundant towards right base. Dulness over lower three-fifths of right back. Fine friction-like but indeterminate crackling sounds below right mamma, where sharp pain is felt on drawing a deep breath. Elevation and expansion of chest-walls both imperfectly accomplished. A roughness, but no definite murmur, with the systolic sound at the heart's base, and along the lower margin of the præcordial space.

Ordered a mixture containing acetate of ammonia, aromatic spirit of ammonia, spirit of nitrous ether, and tincture of squill; simple poultices to chest; and three ounces of brandy daily.

At 9 p.m. the pulse was 108; respirations 36; temperature 101·4°.

On the two ensuing days, May 4th and 5th, the cough was harassing; the expectoration copious, muco-purulent, shreddy, and fetid; the urine acid, depositing lithates, but free from albumen, and of specific gravity 1025. The pulse, respirations, and temperature all rose within this period, and on the evening of the 5th marked respectively 120°, 52°, and 102·1°.

On the 6th of May dry cupping glasses were applied between the shoulders, and a mixture ordered containing senega, carbonate of ammonia, and tincture of squill.

May 7th.—Slept little; respiration rapid and distressing. Lower region of chest on the right side dull as before; but just above the line of dulness the râles yield a marked amphoric note, and the percussion sounds at the spot partake of the same quality. At the right apex, in the erect posture, the resonance is somewhat similar. No splashing on succussion; no fine metallic tinkling; slight hæmoptysis. Mixture to be continued, and dry cupping repeated. Chloral hydrate, fifteen grains, to be administered at night.

8th,—Much relieved by cupping. Slept well. No blood in sputa this morning. Urine of specific gravity 1018, and free from albumen. No trace of amphoric râles to-day, but around right mamma, in the recumbent posture, the percussion note is intensely resonant. Suffers from diarrhœa. Ordered four ounces of brandy and three ounces of sherry daily; compound chalk powder with tincture of catechu, in draught, when requjred. Repeat senega mixture.

9th.—Slept ill. Expectoration more profuse, more puriform and slightly streaked with blood. No amphoric râles, but a cracked-pot sound near right mamma.

10th.—He became drowsy, and in the night restless and somewhat delirious.

12th.—The drowsiness increased, and the breathing grew weaker and more laborious. He now lay for the most part low in bed. Percussion note over right front of the chest unduly clear and ringing.

13th.—Died in convulsions at 11.33 a.m.

At the autopsy, which took place a few hours after death, the thorax alone was allowed to be examined. On removing the right lung the pleural sac was found to contain about two pints of a thin purulent fluid, of intense fetor. At the extremity of what appeared to resemble a small pouch or sinus, retreating from the general cavity of the pleura, a bronchial tube of rather large size opened into the interior of the sac. The sac itself was again subdivided by adhesions into two distinct, but communicating, compartments—an upper and a lower, the upper occupying the space between the lung and the ribs, and the lower intervening between the lung and the diaphragm. The right lung was considerably compressed against the spinal column; there was a general increase of its fibrous tissue, and at the apex a slight amount of consolidation, but no trace of tubercle in any part. At the base of the left lung there was a circumscribed abscess, about the size of a walnut; and above this another, about the size of a hazel-nut. Around these cavities all the tissues were dense and indurated, and throughout the lung-substance there was some fibrous degeneration, but no tubercle anywhere. The mitral valve was thickened, and there was a small fimbriated excrescence upon one of the segments of the aortic valve.

Gentlemen, there are many varieties of pneumothorax as distinguished by their varying modes of origin. Some of these appertain almost exclusively to the surgeon. They originate in penetrating chest-wounds, in abscesses connected with diseased bone, in fractured limbs lacerating the lung-substance, and the like causes; others, again, are mainly remarkable for the extreme rarity of their occurrence—indeed, they are so rare that the majority of you might spend a long lifetime in the exercise of your profession without encountering a single specimen. All these I shall pass over, and confine myself to purely medical pneumothorax, as we meet with it in ordinary practice.

Pneumothorax, then, may originate in empyema of old

standing. Empyema is virtually abscess of the pleural cavity, and, like any other abscess, it will seek to find an exit for its circumscribed pus in one direction or another. It may penetrate outwards through the integuments and reach the external atmospheric air, or it may proceed inwards through the visceral pleura, and come into contact with the internal pulmonary air. In either case there will be pneumothorax superadded to empyema. When the perforation takes its course from without inwards—from the pleural sac into the lung-tissue—you may call the pneumothorax centripetal, regarding the lung as a centre, and the pleural sac as a periphery.

Another variety, by far the commonest of all, is the converse of the last. In the vast majority of instances it originates in the rupture of an intra-pulmonary abscess or vomica into the cavity of the pleura, whatever the nature of the vomica may be. As the course of the perforation here is from within outwards —from the lung as a centre to the pleural sac as a periphery— you may call this variety centrifugal. Pneumothorax of this kind, and that which is due to centripetal perforation in old empyema are the most important varieties that fall within the province of the physician; and to one or other of these two our own case obviously belongs. To which of the two shall we say? It is not easy to answer this question at first sight. There is a difficulty in discovering the precise outline of a vomica *within* the lung-substance at the spot where the rupture took place. Nevertheless, in spite of this difficulty, I believe the rupture to have been centrifugal—that is to say, directed from an intra-pulmonary vomica into the sac of the pleura. My opinion is this. Within some indefinite period before admission, a vomica of moderate size broke through the visceral pleura at once in mass by an aperture of communication as wide as itself, or ultimately formed such aperture. The walls of the vomica then in course of time became almost insensibly merged in the pleural membrane, and the interior of the vomica incorporated with the pleural cavity. The appearances at the aperture are in themselves suggestive of this view, much resembling as they do those of a small secondary pouch engrafted upon a large aneurysm, and the same view is amply corroborated by the existence of vomicæ in the opposite lung. These vomicæ were probably pneumonic in origin. I cannot believe they were pyæmic. You may call them phthisical, if you please, but, in the absence of any existing trace of tubercle, you can hardly call them in any sense tubercular.

There remains yet another problem for solution. Could we

be certain during life that this was a case of pneumothorax? You are aware that the physical signs of pneumothorax are singularly striking—almost unique. I will not enumerate them here. Suffice it to say that the ringing, echoing, reverberating quality of almost every one of these physical signs, when they are widespread and well developed, as they usually are in general pneumothorax or hydro-pneumothorax, is perfectly distinctive, and its evidence unimpeachable. In our case the signs were ill-developed; some of the number, indeed, were absent altogether. Moreover, they were inconstant, many of them disappearing at times. Now, under these circumstances, a question might fairly arise whether we should not ascribe the existing physical signs to the presence of a considerable cavity within the lung substance, immediately contiguous to the chest wall; or, if pneumothorax there needs must be, whether the pneumothorax was not limited in extent, and circumscribed by surrounding adhesions. I am of opinion there was sufficient evidence to enable us with a high degree of probability to decide in favour of general pneumothorax. When the patient reclined on his back, the whole front region of the chest on the corresponding side yielded a clear ringing percussion tone. When he sat upright a resonance of similar quality was elicited extensively over the apex of the lung; there must, therefore, be underneath a large air-containing cavity, and this must almost of necessity imply general or all but general pneumothorax. As a matter of fact, there were adhesions, but these rather distorted the shape than diminished the volume of the pleural cavity.

I have just told you the physical signs of pneumothorax are singularly striking, and almost unique. So also for the most part are the symptoms of pneumothorax. They consist, in one word, of sudden and insufferable dyspnœa, with acute pain, and it may be the sensation of something having given way within the chest. Now, on reviewing the past history of this man, there is not a trace to be found of any such event. Let me offer an explanation of this apparent anomaly. As a rule, in perforation from within outwards, the first occurrence is the entry of air into the pleural sac; the second—the natural result of the first—is pleurisy with effusion, or inflammatory hydrothorax, the two together constituting the compound state known as hydro-pneumothorax. It may happen, however, in a few rare examples, even of centrifugal pneumothorax, that the sequence is reversed. Pleurisy, you know, is common enough where there are vomicæ within the lungs; often dry

pleurisy, sometimes pleurisy with effusion. Now suppose an abundant pleuritic effusion already existing in association with an unsound lung, there is no reason in the world why a pulmonary vomica in the immediate neighbourhood of the pleura should not break through that membrane, and introduce air into the pleural sac. Well, what then? You will not in this case have the sudden and insufferable dyspnœa which overpowers the patient in an ordinary case of pneumothorax, because the lung has already been undergoing compression from day to day, and the machinery of respiration has learnt to accommodate itself to its surroundings. Under these conditions a little air finding its way into an already overloaded pleural sac will be a matter of comparatively trivial moment as far as increase of pressure and dyspnœa are concerned, and need not necessarily be signalized by any symptoms calculated to make an abiding impression upon the memory of the sufferer. Bear in mind, I say the entry of air is of no great moment in the case assumed, as far as increased pressure and dyspnœa are concerned. It may, however, be most momentous in its consequences in so far as they concern the characters of the effusion and the ulterior course of the disease. The effusion, simply sero-fibrinous in the beginning, when incessantly exposed to the damaging influence of air and other matters derived from the vomica, may become puriform and the disease run rapidly from bad to worse. Again, in any case, with or without preceding pleurisy, there may be the same immunity from suffering when the lung-structure is utterly disorganized and well nigh worthless as a respiratory organ. The lung then has nothing to lose by extraneous compression, and where no harm is done, no shock is felt. These last remarks are made by way of parenthesis; the main point to which I invite your attention is that the invasion of pneumothorax, even in its centrifugal form, although usually, is not invariably accompanied by the striking subjective phenomena to which I have alluded.

A few additional points deserve to be commented upon. The bronchial respiration on the left back is abundantly explained by the discovery of vomicæ in the left lung, with indurations around. The alterations in the first sound of the heart—for they were nothing more, certainly not murmurs—are equally well explained by the rudimentary valvular lesions discovered after death. The next point I shall dwell upon at greater length, for it is a matter of practical importance at the bedside. It is noted that on the 12th of May the patient

changed his habitual posture from the erect or semi-erect to the recumbent. Gentlemen, you are all familiar with the fact that there are various modes of dying. One man is said to die from the lungs by apnœa; another from the brain by coma; a third from the heart by syncope or asthenia. You may not be so familiar with the fact that many men die, so to speak, a compound death—by a combination of two or more of the modes of dying enumerated. For example, it is very rare indeed for the victim of old-standing chest disease to die of apnœa alone, although at first sight that might appear to be the sole, or at least the predominant, agency at work. In these cases there are almost always two elements in action—asthenia and apnœa; the two react strongly upon each other in many ways, and conspire together to take the life of the patient. You know perfectly well what is meant by orthopnœa: when the breathing is seriously embarrassed, a man sits upright in bed to save his diaphragm and his lungs. You know what is meant by syncope, faintness, and asthenia: a man lies low in bed to save his heart, and to economize its power of propulsion mechanically. For a long time, in the majority of chest complaints of old standing and great severity, orthopnœa is the rule: the patient sits upright, and can endure no other posture for a permanency; at last, it may be, he begins to lie down persistently, while yet all the signs and symptoms of carbonic-acid-poisoning and malaëration of the blood—with the sole exception of orthopnœa—are as prominent as ever. What does all this mean? Why this: of the two destructive agencies at work—debility and dyspnœa, or rather debility and malaëration of the blood—debility is now in the ascendant. It means, moreover, that the blood-contamination, once so strongly resented by the respiratory organs in the form of dyspnœa, is at last, amid the universal apathy and prostration, so to speak, either unfelt or disregarded. The entire system of organic life seems paralysed; the necessity of breathing makes no impression upon it, and provokes no salutary response or reaction. Such a state of things, you may readily imagine, is imminently perilous to life; it is, in truth, the beginning of the end.

It is deeply to be deplored that, while pages have been written on the genesis of pneumothorax and on its characteristic signs and symptoms, half a dozen sentences have too often sufficed for the description of its treatment. Nothing can be more dreary and disheartening than the brief commentaries of the text-books on this point. The burden of the song is the same in all. At the onset administer opium to assuage pain, give

ether, ammonia, and other antispasmodics to control dyspnœa, and, *if need be*, as an extreme measure, take blood from the arm or apply the cupping glasses in order to save the opposite lung from the accession of collateral hyperæmia and œdema. During the progress of the case, sustain the strength, palliate untoward symptoms as they arise, and reserve paracentesis for an emergency—at least, in cases of consumption. If this is all we can do, it is a lame and impotent conclusion indeed, a melancholy avowal of the shortcomings of science. Perhaps, in a few particular instances, bolder measures might be allowed without reference to routine. One thing seems to be omitted by most systematic writers on chest disease, and I myself plead guilty to its omission in practice. It is the bounden duty of all physicians in cases of pneumothorax with effusion, to explore the chest from time to time, and to determine the nature of the fluid effused, at all events, in cases where fever is present. Pus there may, and probably will be, and opinions may differ as to the proper way of dealing with a laudable empyema, whether accompanied or unaccompanied by air in the pleural cavity, but foul, unwholesome pus is another affair, and must be withdrawn at all hazards. Hence the absolute necessity of exploration in all febrile cases. It is vain to say that the constitutional symptoms will give you fair note of warning, and tell the truth in due season; that the quick and fluttering pulse, the vacillating temperature, and, it may be, the occurrence of shivering, sweating, and delirium, will set the question at rest, and establish the existence of putrid pus beyond the shadow of a doubt. I assure you, gentlemen, if you wait for all, or even any of these things, you are greatly to blame. If your patient is fairly calm and comfortable, you may let him alone, but in the midst of fever, dyspnœa, and distress, it is impossible to draw fine distinctions, and to await the mere aggravation of pre-existing symptoms, for, after all the question is in many cases one of degree only. Again, I say I am myself open to my own rebuke, and all the more open to it, inasmuch as in a case of pneumothorax the loose puriform fetid sputa pointed naturally to an overflow from the pleural cavity.

Another point insufficiently dwelt upon is the prophylaxis. I am sure there are certain cases of phthisis which present good grounds for the suspicion of coming pneumothorax. Take, for example, the following case. It is no imaginary picture, it is drawn from the life within my own experience. A patient lies steadfastly on one side, and dare not recline on the other for fear of provoking pain, irritation, and cough. As a passing

occasional phenomenon this is a matter of no great moment, but let this state of things last, as I have known it to last, uninterrupted in time, and unmitigated in degree, for many weeks in succession, then it assumes a different aspect and a deeper significance. Clearly, under these circumstances, there must be something serious to account for the sustained irritability of the pleura, and in far-gone phthisis the *fons et origo mali* must almost of necessity be the presence of vomicæ underneath the pleura, and, I may add, underneath a part of the pleura unadherent and unprotected by false membrane, for adhesions and false membranes would inevitably bar all severe and continuous pain at the seat of their formation. Such cases are at any instant exposed to the risk of pneumothorax.

If, then, we are fairly forewarned of the peril in prospect, our course of action is clear. Rest and support to the system are of course essential, and so is strapping of the side, but, above all, we are bound with the aid of anodynes and emollients to keep down the characteristic cough of phthisis, a form of cough at once exhausting and in great part superfluous; not required, I mean, for any work it has to do in the way of expectoration; in a word, a form of cough which is imminently dangerous from the strain it puts upon the damaged lungs, and upon their frail investing membranes.

LECTURE VII

ON A CASE OF PNEUMOTHORAX

Delivered March 22nd, 1872. Published in 'British Medical Journal,' October 5th, 1872.

John B—w, æt. 22, was admitted originally on the 12th of August, 1871, under Dr. Cayley, to whom I am indebted for the preliminary notes up to the date of his discharge in October. His own antecedents and his family history were good; his appearance was that of a healthy and well-made man.

On admission, the heart was found beating most distinctly in the third right interspace, just beyond the sternum; there was considerable apparent bulging on the left side, which was dull from apex to base, and failed to expand on deep inspiration. The breath-sound was feeble everywhere, quite inaudible towards the base of the lung, and faintly blowing at the front apex.

August 17th.—Paracentesis was performed by Mr. Hulke between the sixth and seventh ribs, and ninety-eight ounces of clear serum of specific gravity 1020, were withdrawn. During the operation, the heart returned towards its normal position.

21st.—There was fair resonance in front as far as the fourth rib; in the axilla, to the level of the fifth; and at the back, as far as the angle of the scapula.

September 16th.—Friction-sounds were audible over the left front. There was dulness anteriorly below the fourth rib, and posteriorly from the base of the lung up to the middle of the scapula. Over the dull space, the breathing was weak and bronchial; and there was an ægophonic twang with the voice.

October 3rd.—The patient left the hospital for Eastbourne without any signs of pneumothorax.

23rd.—He returned from Eastbourne. He never lost his dyspnœa entirely. About Christmas last, he had a "bilious attack," accompanied by much retching. Shortly afterwards, the dyspnœa became more distressing, and the "washing-sound" described in the following notes began to be heard. On the 23rd of January, 1872, he was again admitted, and placed under my care.

On re-admission, little or no breath-sound could be heard over any part of the chest on the left side. Over the upper three-fifths of the same side posteriorly the percussion-note was clear, though short; in front it was inordinately clear and full. At the posterior base there was absolute dulness, and, in the same region, splashing and tinkling

sounds might be elicited on succussion. The heart's impulse was most distinctly seen and felt at the midsternum and in the third right interspace, and again it was well perceived at the ensiform cartilage. The heart-sounds were widely diffused over the front of the thorax on both sides of the sternum, but they were more plainly audible on the right side.

In the above description, reference is made to the ordinary postures of the patient during examination; when he changed his posture, the limits of dulness and resonance at once interchanged places.

He was himself quite conscious of the splash within his chest whenever he moved, and in particular when he rocked himself; he called it a "washing sound," and was much annoyed by it. He was able to walk a short distance without distress, but was incapable of prolonged exercise. Ordered a draught containing aromatic spirit of ammonia, spirit of ether, and syrup of tolu every four hours; with four ounces of port wine daily.

January 24th.—No albuminuria. Dulness over the main seat of the heart's impulse in the third right interspace, extending an inch and a half beyond the margin of the sternum.

26th.—Measurements of chest at the nipples, right side, $17\frac{5}{8}$ inches; left, $18\frac{3}{8}$. With two florin pieces, the bell-sound may be easily demonstrated on the resonant surface of the left side.

31st.—Metallic tinkling heard in unison with the movements of the heart.

February 3rd.—Over the right supraspinous fossa, the breathing is bronchial, and there are creaking sounds of indeterminate character. Respiration audible, though feebly, over nearly the whole of the left back.

8th.—Up to this date, the pulse ranged from 88 to 120, and the temperature from 97° to 100·8°. These were extremes, the maximum of the pulse-rate and the minimum degree of the thermometer occurring only once. The prevalent average was, for the pulse, about 99; for the temperature, about 100°; for the respirations, 27. On this day, paracentesis thoracis was performed by Mr. Hulke; and thirty-six ounces of greenish-yellow inoffensive pus were withdrawn bodily by an aspirator, and an indefinite amount afterwards removed by washing the pleura with water—probably about twelve or thirteen ounces. Along with the pus, bubbles of air were seen passing through the cylinder of the aspirator. During the operation, the heart's dulness and impulse were perceived to subside downwards and leftwards, and in the end they reached nearly the normal position, where the heart-sounds were now more distinctly audible than on the right side of the sternum. The measurements were, right side, $19\frac{5}{8}$ inches; left, $17\frac{3}{8}$.

9th.—Patient states that on moving in bed he no longer hears the washing, but on succussion the tinkling is still perceptible. Gurgling râles of high pitch and ringing tone distinctly heard over the upper part of the left front. Posteriorly on the left side the breathing is audible down to the level of two inches above the extreme base, where there is dulness on percussion.

10th.—Expansion of left side almost *nil*. Heart's impulse conspicuously visible over the sternum opposite the nipple.

11th.—There is pain in the right hypochondrium when he lies on the left side. Shivered slightly in the afternoon, and in the evening there was a marked rise in the pulse and temperature.

12th.—Passed a very restless night. On taking a deep breath he complains of pain beneath the right mamma where friction-sounds may be heard. There is dulness below the third right cartilage for the space of an inch from the margin of the sternum. Ordered a pill composed of opium, quinine, and digitalis. Ether and ammonia draught to be repeated. Dry cupping to the painful part of the right side.

13th.—He is said to have "vomited" this morning a quantity of greenish-looking pultaceous matter.

16th.—Pain of an aching character along left clavicle. Pulse small, compressible, and undulating. Ordered six ounces of port wine daily.

19th.—Prolonged expiratory murmur at right apex. At right base rhonchus anteriorly, harsh breathing posteriorly. Heart-sounds louder to the right of the sternum than to the left. Moist râles under both clavicles. Pulse full, feeble, tremulous and jerking. Ordered four ounces of port wine and four of brandy daily.

23rd.—Sputa very abundant and most offensive to the taste. They are expectorated gently in small quantities at a time, and consist of muco-purulent matter in which pus largely predominates.

26th.—The porringer has been nearly filled since last evening with greenish-yellow sputa, much resembling the fluid withdrawn in the operation of the 8th.

29th.—Looks pale with a slight flush. Finger-nails beginning to be curved. Continued profuse expectoration of pus. Sonoro-sibilant râles over right back; sounds of mixed creaking, gurgling, and sucking characters under the left clavicle.

March 5th.—Expectorates most abundantly and most easily in the morning with a slight cough in the first instance without apparent straining of any kind. Perspired copiously during the night. He is now greatly emaciated.

7th.—Night sweats held in check by zinc and henbane.

9th.—Friction-sounds heard almost universally throughout the right side; at the front apex they are replaced by bubbling râles. Sputa unchanged in all respects.

From February 8th up to this day, the pulse has oscillated between 90 and 138, the respirations between 24 and 36, and the temperature between 99° and 103·6°. This last reading, however, was only once noted. As a rule, the temperature rarely exceeded 102·6°; and there were not more than two or three occasions on which the pulse-rate fell below 100, or rose above 130.

11th.—Pulse, 144; respirations, 49; temperature, 103·2°. Loud, coarse, characteristic friction could be heard and felt over the lower two-thirds of the right front, and over a corresponding area towards the right posterior base. The patient declared that he himself felt the rubbing very distinctly when he placed his hand on the right side of the chest. Pus, as before, was expectorated in large amount. He was ordered a pill contaning two grains of quinine, one grain of sulphate of iron, and a quarter of a grain of opium, to be taken three times daily. At 10 p.m., the patient was much distressed by the incessant outpouring of sputa. About this time, the exhaustion was very great, and the cough began to slacken in force.

12th.—At 4 a.m., the cough, expectoration, and outflow of pus, all ceased entirely; the breathing became hurried; and there were loud liquid rattles in the throat with each inspiratory act. Respirations 68. Between 9 and 10 a.m., on being raised, the patient seemed to breathe

more easily for a few minutes, when his head fell back, and he became livid, and died in a quarter of an hour afterwards.

Necropsy, twenty-four hours after death. For the particulars, I am in the main indebted to Dr Robert King. *Thorax.* On opening the chest, there was no rush of air from either pleural cavity. The pericardium, with the heart and left lung, were pushed over to the right, away from the side walls of the chest. The lung, however, was firmly attached by its greatly thickened pleura and by fibrous bands to the apex of the pleural cavity, along the left edge of the upper half of the sternum, and, through the medium of two tail-like prolongations, to the diaphragm. The lung-mass itself was not much larger or thicker than a man's hand; its lower two-thirds being of a dark slate colour, and completely collapsed; while its upper third was riddled with large irregular cavities, which freely communicated with each other, and by three or four openings with the pleural sac. The widest of these orifices would admit the tip of the little finger. The pleural sac itself was nearly filled with thin watery pus, and more than six pints were removed. On opening the trachea, the lung remaining *in situ*, water could readily be made to flow through it along the left bronchus and through the aforementioned vomicæ at the apex into the cavity of the pleura. On making sections of the luug, a few old cheesy deposits were observed; but no trace of miliary tubercle could be found. The parietal layer of the pleura was thickly covered with corrugated masses of lymph, which, along the crests of some of the larger rugæ, were fully an inch and a half in thickness. The *right lung* was coated with recent lymph, especially over its lower lobe and between its base and the diaphragm. This lower lobe was everywhere intensely congested, and scattered freely throughout its substance were numerous patches of lobular pneumonia. The upper portion of the lung was densely indurated, apparently by the coalescence of a number of patches similar to the last in origin, but differing in the greater amount of caseous change which they exhibited. In two places, the caseous material had broken down, and left considerable cavities. No unequivocal example of gray miliary tubercle was discovered anywhere. The upper division of the *left* bronchus was traced continuously into the anfractuous cavern at the apex before described. Having been washed with water from the trachea before it was opened, no pus was discovered in it. The right lung, however, contained pus, along with frothy mucus, in its bronchial tubes. The bronchial membrane in both lungs was deeply injected. The vocal chords were immensely thickened. The heart was to all appearance healthy, its valves being competent and free from deposit. There was no excess of fluid in the pericardium.—*Abdomen.*—The liver especially its right lobe, was attached by recently organized lymph to the under surface of the diaphragm. Glisson's capsule was minutely studded with gray miliary granulations, perfectly distinct, nowhere coalescing, of a definite rounded form, almost transparent, and nearly uniform in size, the largest being scarcely bigger than a good-sized pin's head. The substance of the organ was fatty and somewhat friable; it contained no visible trace of tubercle. The kidneys appeared healthy, and their capsules were non-adherent; they presented no tubercle. The spleen, on section, showed numerous whitish points, whether they were Malpighian corpuscles or true miliary tubercles. The omentum and mesentery were thickly studded with tubercle throughout; and the intestine on its peritoneal surface—more particu-

larly the ileum—was beset with similar granulations, which occurred in patches corresponding to irregular and dirty-looking ulcers on the inner surface of the bowel. These ulcers were met with throughout the large intestine as low down as the rectum, were most numerous at the lower end of the ileum, and extended quite to its upper extremity. They were not found, however, in the jejunum or duodenum.

Gentlemen,—You have in the case just detailed a good example of hydropneumothorax, with most of the characteristic signs fairly developed.

The question naturally arose as to the existence of phthisis. I had no reason, judging from the history and from the physical examination of the chest, to suspect the presence of advanced phthisis; and in some degree advanced it must be in order to perforate the pleura through the medium of a vomica. I had no reason to feel assured that there was any channel of communication still patent between the bronchial tubes and the pleural cavity; for aught I knew to the contrary the orifice might be closed, and well closed after the lapse of so long a time. Under the circumstances assuming the absence of the above pathological states and knowing that the man was feverish and disabled, incapable of work, and even of ordinary exercise—knowing also, or fearing at least, that without surgical aid he must be doomed unceasingly to hear the worrying splash within his chest, and that sooner or later in all likelihood his health and strength must give way—I thought myself warranted in submitting him to the operation described in the notes. For a day or two, the result was all that could be desired. A small residue of pus, it is true, and a considerable volume of air, still remained; but the breath-sound was heard more freely and extensively over the upper region of the lung, and the heart had regained nearly, if not entirely, its normal position. Soon there ensued a period characterized by shivering, fever, vomiting, and pain in the right side of the thorax. In a word, he was suffering from an attack of pleurisy on the side hitherto comparatively unaffected. Fortunately, this attack was unaccompanied by any large amount of liquid effusion, or we might have been compelled, under peril of immediate death to the patient, to tap the right pleural cavity within a few days after the original operation on the left side. Unfortunately, along with the pleurisy, so suspicious in itself, a number of new and alarming signs and symptoms began to declare themselves, and to assume prominence. Sounds of grave significance were persistently

heard under both clavicles; and, although in the beginning we indulged the hope that they might only be pleuritic friction-sounds on one side, and on the other intrabronchial *râles* more or less modified by the neighbourhood of a resounding cavity, and that all might disappear in time, we found that, although they changed, they did not disappear, but remained, obviously suggesting the idea of confirmed pulmonary phthisis. In the meanwhile, pus issued freely and almost spontaneously with the sputa; the left side rapidly refilled; and the heart again occupied its old place beneath the sternum and to the right of that bone. The characters of the pulse were of the worst omen; there were night-sweats and emaciation; and the fever degenerated into well pronounced hectic.

We were now in a position to give a fair interpretation of the case, and it appeared almost impossible to resist the following conclusions. The man was originally phthisical, although for many months the true state of the lungs escaped observation. In August last, he was tapped for simple pleurisy with effusion; a certain amount of fluid remained after the operation, and increased until the beginning of October, the date of his discharge. Shortly after the ensuing Christmas, a vomica at the apex of the left lung broke through the pleura, and introduced air into the pleural cavity, converting the simple sero-fibrinous effusion into pus, and establishing empyema with pneumothorax. I will not stop to inquire what were the agencies at work in the process of conversion: whether the perpetual influx of air was alone sufficient to accomplish the change, or whether it was in great measure owing to the simultaneous introduction of puriform and putrid materials from the vomica—an event which must surely take place in phthisical pneumothorax, although its possible influence has been strangely overlooked. For a while, the broncho-pleural fistula remains open; at length it closes, or, if unclosed, it is a mere valvular slit in the pleura. In this state the patient comes under my charge. Ultimately after the operation, when the disembarrassed lung recovers some portion of its lost respiratory power, and, I may add, when the forces of destruction at work within the tissues are set free from the pressure that held them in check, the closed orifice reopens or the valvular slit becomes a wide-open gap.

The *post mortem* appearances were in the highest degree satisfactory, in so far as they verified to the letter the interpretation we had put upon the case. I will not dwell at

length upon the immunity of the lungs from tuberculization, so strikingly contrasted with the presence of tubercle elsewhere, however interesting this may be from a pathological point of view. It is true that, even practically regarded, the possible development of tubercle, as a result of infective processes, is a matter of serious moment when we are deliberating on the treatment of a pleuritic effusion which may be, or may become, an empyema. In our case, however, the tubercles were unimportant, and had nothing whatever to do with the fatal isssue. The points of real importance were the disorganization of the right lung, the quantity of pus in the pleural cavity, and the wide, straight, unbroken channel of communication between that cavity and the trachea. And now that we are in possession of all the facts—the disclosures of the dead-house, and the history and progress of the case from beginning to end—dare we venture to affirm that we could have prolonged the life of this man for a few days or weeks? I cannot say more: he was too far gone for a new lease of life. As a preliminary to the solution of this problem, let us examine the mode of dying. An immense amount of pus had been long as it were overflowing in successive gushes through an open and capacious broncho-pleural fistula. Now reflect for a moment on the mobility and incoherence of pus pure and simple, as distinguished from the comparative viscidity of sputa in general, and you will be prepared to acknowledge that there is reason in the following remarks. With declining strength, with sharp pleurisy and a damaged lung on the right side, with a feeble cough and failing powers of expectoration, it became almost inevitable that a portion of the outgoing pus from the left pleural sac must find its way backwards—regurgitate, so to speak, through the right bronchus into the corresponding lung, the only lung on which devolved the duty of such expectoration as could be accomplished by lung-tissue under the circumstances. For some time the right lung continues to perform this duty, resents the invasion of pus, and eliminates it; but all the while, of course, it is suffering from irritation; and in the end the irritation becomes inflammation, or quickens pre-existing inflammation. The elasticity of the lung is profoundly impaired, and its intrinsic powers of elimination are reduced to a minimum. Empyema with pneumothorax on one side, and extensive phthisical pneumonia on the other, must of necessity prove fatal in a short time, even without the oozing of pus into the trachea, or its regurgitation through the opposite bronchus. *With* this oozing, and *with* this regurgita-

tion, you may easily understand how it might prove immediately fatal. The least movement of the chest-walls, the slightest change of posture, propels the fluid into the trachea, where part remains, while part regurgitates; a far stronger movement is demanded for its expulsion; under the co-operating influences of exhaustion and inflammation, that movement is not forthcoming, and the patient dies asphyxiated. This is the great danger we have to dread. Can we obviate it? *As a matter of fact* now that we are enabled to read the case backwards by the reflected light of the issue, I believe the best thing we could have done was to have inserted the drainage-tubes in the first instance after the operation in February. We were not then, however, in possession of all the data; the pus withdrawn was inoffensive, and as yet there was no overflow from the pleural cavity. Accordingly it was determined to close the wound, and reserve the chest for a future operation, if necessary. The question now arises, Was there a necessity for a second operation? In my own opinion, there was. The second operation, however, should not have been delayed until the pleural cavity became brimful of pus; it should have been performed, and the drainage-tubes inserted, as soon as there was evidence that the pus, though evacuated in immense amount, was steadily gaining ground, while the air was fast disappearing.

I have dwelt in detail upon this case, inasmuch as it offered from the beginning, and even yet offers, the amplest scope for speculation and discussion. Moreover, cases of pneumothorax are few and far between even in the large experiences of hospital practice; and this particular case requires in great measure to be estimated on its own merits. You will derive little advantage in the solution of the problems propounded by consulting the text-books. For the most part, they content themselves with saying that, in pneumothorax with phthisis, paracentesis should be reserved for an emergency. By emergency is meant in all probability the *existence* of dangerous dyspnœa, septic poisoning, or severe distress. It is not so certain that the *prospect* of danger ahead is contemplated under the term, but I am convinced that even prospectively regarded the operation may be justifiable.

To recapitulate and to conclude, I am of opinion that in the first instance when the hydropneumothorax had existed already for three months or more, and there appeared to be no chance of a spontaneous cure—under the circumstances and with the lights we then possessed—an operation was unavoidable.

Whether as *a matter of principle* under the same circumstances the drainage-tubes should have been inserted at once, may be open to question; but most undeniably they ought to have been inserted in the progress of the case after the discovery of the following conditions: free and direct communication between the left pleural cavity and the trachea, advancing disorganization of the right lung, and ever-increasing accumulation of pus, in spite of its enormous discharge. By adopting these measures, we might have kept the pus within bounds, and diverted it from the trachea and bronchi; we might have arrested for a while those pneumonic processes which were so rapidly disabling the only lung of any real use in respiration; and, although the man must inevitably have died an early death from combined exhaustion and apnœa, we might at least have saved him from dying of downright suffocation—literally drowned in pus.

LECTURE VIII

ON A CASE OF HEART DISEASE, OLD AND RECENT

Delivered January 8th, 1875.

William E. K—, æt. 12, born of a rheumatic mother, was admitted November 11th, 1874.

On the 21st of October, he began to suffer pains in the ankles and knees, and two days afterwards in the shoulders; the pain was severe, but, according to his own account, it was wholly unaccompanied by redness or swelling. About the 3rd of November he was taken with a cough: on the 7th of November he felt some pain in the left side of the chest, but neither then nor at any time until the day of admission, did he experience any shortness of breath.

On admission, pulse 132; respirations 52; temperature, 100·5°. Breathing universally high-toned, and harsh. At both posterior bases it is distinctly tubular, more extensively at the right base, and over the same regions there is either dulness or impaired resonance on percussion. A systolic murmur at the heart's apex. Ordered a mixture containing acetate and carbonate of ammonia, and a linctus containing morphia. A belladonna and iodide of potassium ointment to be applied to the præcordia. Brandy, two ounces daily.

November 12th.—Urine of specific gravity 1022; yields albumen, one twentieth, but no chlorides. Pulse 148; respirations 60; temperature 102°. Voice-sound tremulous, and twanging on both sides of the chest, but more markedly on the right. Cough short and harassing. Friction-sounds may be heard over the præcordia. Ordered a mixture containing acetate of ammonia, aromatic spirit of ammonia, tincture of squill and compound tincture of cinchona every four hours.

13th.—Pulse 126; respirations 48; temperature 100·8°. Præcordial dulness increased in area. Pain between heart's apex and ensiform cartilage. A leech to the painful part.

14th.—Pain relieved at the præcordia, where, however, the dulness is still extensive, tapering on both sides of the chest to the second cartilages. Tubular breathing well pronounced over the upper two thirds of the left back and over the lower two thirds of the right; but in point of fact the breath-sounds are more or less of this character everywhere over the right side posteriorly. An occasional transitory half-musical sound may be heard over the præcordia with the heart's systole.

15th.—Pain near the left nipple on deep inspiration and on coughing. Fine crepitus at both posterior bases.

16th.—No albuminuria.

19th.—Pulse 90; respirations 32; temperature 100°. Dulness and

distinct tubular breathing over the lower half of the right back, but hardly elsewhere—only an approach to the tubular type over the left upper lobe.

21st.—Pulse 104; respirations 27; temperature 99·6°. Pericarditic rhythm well marked. Præcordial dulness diminishing in area.

26th.—Double murmur in carotids, not, however, wholly effacing the second sound. Same at the base of the heart,

27th.—Præcordial dulness lessening in area.

30th.—Severe pain, increased by deep inspiration, at the margin of the left lower ribs. Constant hacking cough. Harsh tubular breathing over the greater part of the right back, which is more extensively dull on percussion. A leech to the painful part. Sherry, six ounces daily. Compound ether and ammonia draught when required. Mixture to be repeated every four hours. At 8 p.m., respirations 50; pulse 168. Four ounces of brandy ordered. Leech-bite bled very profusely; bleeding stopped by tincture of perchloride of iron. He coughs less, but breathes more laboriously. At 9 p.m., pulse 154; respirations 60; temperature 101°. Sputa scanty and tenacious, but not rust-coloured; breathing short; face flushed. Perspires freely. At 9.45 p.m. he died.

Autopsy.—After removal of the sternum and rib cartilages, the much enlarged heart was seen to occupy nearly the whole of the space brought into view. Numerous adhesions, readily broken down, existed between the left lung and the parietal and diaphragmatic pleuræ, while a few ounces of blood were contained in the corresponding sac. The right lung was free from adhesions, but the pleural cavity on that side contained nearly one pint of dark straw-coloured fluid without much, if any, admixture of lymph. The upper lobe of the right lung was crepitant, though somewhat congested. Middle and lower lobes smooth on surface and of a purple colour; on section they exhibited a uniform dull-red appearance; they were firm to the touch, sank in water, and exuded a large quantity of thin airless fluid on pressure. The bronchi within the lung were inflamed and filled with frothy secretion. Left lung congested and œdematous; in other respects healthy, save that the outer portion of the lower lobe, for the depth of about half an inch, presented the same characters of carnification and splenization as were present throughout the whole of the right lower and middle lobes. Bronchi as described in the right lung. The heart was in its greater extent bound down to the parietal pericardium by semi-fibrous adhesions, easily broken through; their bands were chiefly limited to the anterior surface of the heart, for posteriorly the two pericardial laminæ were separated by a small quantity of opaque fluid, the membrane itself being coated by a thick, rough, vascular layer of lymph. The left chambers of the heart were especially enlarged. The right auricle was dilated, but the cavity of the right ventricle appeared to have its normal dimensions. The tricuspid valve, however, was incompetent, though healthy in appearance. On the left side there was incompetency of the mitral and aortic valves. The mitral valve was thickly covered on its auricular aspect by warty vegetations. The endocardium of the left auricle was thickened. The cavity of the left ventricle was large and its walls thick, pale, and soft. The central curtain of the aortic valve was shortened, its border thick and rounded, and the seat of vegetations. Vegetations, however, were most abundantly massed on the right and left segments.

The whole of the liver was seen to project below the ribs, its lower margin reaching to the umbilicus. It was somewhat congested. The

kidneys also were slightly congested. The remaining viscera were healthy.

Gentlemen,—You are all aware that the liability to endocarditis and pericarditis in rheumatism is inversely as the age of a man. That is the main proposition, but there are one or two corollaries to the proposition which may not be so well known to you. In the first place, in young people it is not always acute or even subacute rheumatism that gives origin to heart-disease; in them the faintest trace of articular ailment, the merest modicum of rheumatism, may inaugurate in the heart changes that sooner or later may issue in disorganization and death. The younger the patient the less the amount of the rheumatic poison that is required to determine those changes. A child may have a few fleeting pains in the limbs, and may keep his bed or his room for a few days, and then make a good recovery, to all appearance, in the eyes of his friends, but not so, peradventure, in the judgment of the physician, who may know perfectly well that he bears a damaged heart and a doomed life. In the borrowed words of Corvisart, "Hæret lateri lethalis arundo."

The second corollary from the main proposition is the following. In any young person who is suffering from long-established heart-disease, and who has suffered of late in any degree from attacks of rheumatism unrecognized before, it is fair to presume, all negations notwithstanding, that there may have been in early childhood some fugitive and forgotten attack, which has left an abiding impression upon the heart, if not upon the memory. This conclusion I drew in the case of a girl I lectured upon in 1873, although her known experiences of rheumatism were altogether insignificant and limited to the preceding six months, and to the same conclusion I am irresistibly drawn in the case of this boy, in whom the first ascertained attack dated from three weeks only before admission. In both the one and the other there was a large amount of unsoundness in the heart of old standing, and taking origin clearly from a time long prior to the period of acknowledged rheumatism. The corollaries aforesaid rest, indeed, on wholly different bases, but they are both undeniably true. The first of these, the ascription of heart-disease to a mere trace of rheumatism in early life, is amply verified by all experience. The second, the presumption of a bygone and forgotten attack of rheumatism in childhood under the given circumstances, stands to reason so steadfastly that it is almost impossible to gainsay it, although, of course, it is absolutely impossible to prove it.

I need not again enter at length into the particulars of the heart's unsoundness. Suffice it to say that the hypertrophy and dilatation were of old date; the valvular changes were partly old and partly new, an overgrowth, in fact, of recent excrescences engrafted on the products of past inflammation. The changes in the pericardium must be new altogether, and comprised within the period of the preceding three weeks. All the changes were well attested by their proper evidences during life, but, if you please, I will limit myself to the consideration of two of the physical signs recorded in the notes; one, the pericarditic rhythm, as it is there called, the other, the double murmur in the carotid arteries.

The rhythm of the auscultatory signs in pericarditis—at all events in a very large proportion of cases—is peculiar and all but distinctive. It may be described as a cantering* tripartite rhythm, governing alike the natural sounds when they exist alone, the exocardial murmur when it exists alone, and the combination of murmur and natural sounds when they coexist. The essential element in the rhythm is a doubling of the diastolic sound, or a doubling of the diastolic rub, or the association of both phenomena in one and the same case. Always valuable in diagnosis, the rhythm is of especial value when unassociated with rub. In this simple form it is often in advance of all the signs and symptoms of mischief at work within the pericardium. Whenever, therefore, in the course of rheumatic fever you meet with the rudimentary rhythm I have just defined, you are bound practically to assume the presence of pericarditis, and to act upon that assumption at once without waiting for the development of the authentic and recognized rub. It remains only for me to warn you that when the developed friction-sounds are unusually harsh and grating the rhythm may be lost in the noise and dissonance. Again, in rare cases, where the tones of friction are softer and more subdued, there *may* be no rhythm from beginning to end. The rhythm is only a general rule, it is not a universal law.

The second point for description is the double murmur in the carotid artery. Auscultation of the carotid is mainly useful in affording a measure of injury done to the aortic valve—in the

* We owe the term "cantering" to the late Dr Hyde Salter. I cannot, however, accept Dr Salter's analysis of the rhythm in relation to the several stages of the heart's cycle; at all events it will not accord with the characteristic rhythm. The triple rhythm of pericarditis is discussed more at length in Lecture XI of this volume, with some amount of unavoidable repetition.

language of chemistry, as a quantitative test. Of course, like all quantitative tests, it is also qualitative, determining the nature of the damage as well as the amount; and in this respect it may have its value for an inexperienced auscultator, when, for example, he cannot tell whether the sounds heard at the præcordia point to a local pericarditis limited to the neighbourhood of the great vessels, or to aortic valvular incompetency; or when, given pre-existing pericarditis, he wishes to ascertain whether there may not be superadded incompetency of the valve. For similar purposes he may find it of advantage to consult the carotid artery in some cases of mitral stenosis. Nay, even the best ear may be baffled at the præcordia by emphysema and bronchitis, and the best auscultator may be fain to get rid of the uproar and the interference by appealing at once to the carotid for the diagnosis of the disease. Possibly, also, the test may be available in this respect on a few other occasions, but in the main, I repeat, auscultation of the carotid is a quantitative test with a view to assessment of damages. Let us assume that the natural second sound is generated by the closure of the semilunar valves, without entering into the question how far it is so generated directly by the mere collision of the valve-curtains, how far indirectly by the shock of the recoiling blood that follows the closure. Either way the closure gives origin to the sound. Suppose, now, that you hear about or above the base of the heart a double murmur—systolic and diastolic, for it is seldom diastolic alone—and that you pronounce a verdict of aortic valvular inadequacy; you know that the valve is damaged; you wish to know how far it is damaged. If you fail to hear the second sound at the præcordia, you may at once unhesitatingly conclude that the damage is serious. If, however, you hear the second sound there, followed by a murmur, and, it may be, veiled but not abolished by it, then you are in a dilemma. You cannot decide with unerring certainty whether the second sound is generated by both the semilunar valves acting together, or by the pulmonic valve alone. You may, indeed, guess that the second sound is owing simply to the closure of the pulmonic valve, if it be limited to the left side of the sternum, but it is not always so limited. You proceed then to exclude the pulmonic valve by interrogating the carotid artery; whatever positive or negative evidence you gather by auscultation there, whatever there you hear or fail to hear, so far as the valves are concerned, must all be ascribed to the aorta alone; the pulmonary artery is out of court altogether. What do you hear? In the vast

majority of cases you will hear a murmur with the systole of the ventricle; during the diastole in the first place you may hear nothing at all—neither natural sound nor murmur; or you may hear a murmur alone; and these results of auscultation, mere murmur and absolute silence, would appear to alternate with each other in the most inexplicable fashion. In the selfsame individual, one day you discover the one, and another day the other; nay, more, at the same moment you find the one in the right carotid and the other in the left. More than that, in the same carotid you meet with the one in the upper half, the other in the lower half of the vessel. In the second place the natural diastolic sound may be veiled without being concealed by a murmur, or veiled and followed by a murmur, or simply followed by one. In the last place, when the lesion is slight but sufficient to cause abiding incompetency, a murmur continuously audible at the præcordia may at times be lost altogether in the carotid, and the pure diastolic sound alone remain. Again, you *may* have at times the pure unmodified diastolic sound in yet another class of cases, where the lesion is quite insignificant and the valve is competent by fits and starts, varying intermittently with the varying blood-pressure at the commencement of the aorta. I have thought it my duty to lay before you these particulars, which, perhaps, you may regard as unnecessary hair-splitting, unworthy of a place in your recollection. I care not if you do forget the particulars so long as you remember the main principle that presides over their interpretation. It is intelligible enough and easily enough borne in mind. Let there be known aortic valvular insufficiency, and let the amount of injury incurred be unknown, the presence of the second sound in the carotid is of good omen, its absence of evil import.

Among the physical signs of pulmonary disease tubular breathing occupied a prominent place. Probably it owned a complex origin, resulting to some degree from the pressure of an enlarged heart and a loaded pericardium, but principally from intense œdema, from pleurisy and hydrothorax, and from pneumonia, although it must be admitted that at the time of death the pneumonia failed to present the granular character, if, indeed, it ever had that character at all.

The death of this boy was distressing in the extreme. Up to the 29th of November he had improved wonderfully in all possible respects; the pneumonia, the pulmonary œdema, the pleurisy, and the pericarditis, all appeared to be on the decline, and everything seemingly offered the fairest prospect of re-

covery, in so far as the victim of inveterate heart-disease can ever be said to recover. On the following day the whole scene changed. The boy was seized with a sharp attack of inflammation in the left diaphragmatic pleura, the fluid rapidly reaccumulated in the right pleural cavity, and the dyspnœa became insupportable. A leech applied to the left side gave rise to excessive hæmorrhage, controlled with the greatest difficulty. All these untoward events occurring at once were more than the boy could bear, and he died. Could anything have been done at the last moment to save life, or postpone the hour of death? Ought I to have sanctioned paracentesis of the right side? Perhaps I ought, and possibly I may resort to this step hereafter on an emergency, but except as a folorn hope I should be afraid to do so in heart disease. At the post-mortem examination there were few or no inflammatory appearances in the effusion into the right pleural cavity, nor were there any vestiges of inflammation upon the pleura itself on that side. The fluid, therefore, in all probability, was in the main that of hydrothorax or dropsy of the pleural sac. If so, it would have returned at once after removal, and the relief would have been little more than a momentary respite. On the other hand, the boy might have died under the operation.

LECTURE IX

ON A CASE OF PYÆMIA, WITH VALVULAR DISEASE OF THE HEART

Delivered May 2nd, 1879. Published in 'Medical Times and Gazette,' September 16th, 1879.

William S—, æt. 56, was admitted into the Middlesex Hospital, March 22nd, 1879, when the following notes were taken:—He is unable to give any definite account of his illness. All he can say is that he has had pains in the ankles for the last two or three weeks. Some years ago he was in the surgical wards, under the care of Mr. Nunn, with what he calls erysipelas and consumption, which probably means scrofulous disease of the bones. On his own showing he is "moderately abstemious," and never had syphilis. His general aspect is that of a pale and somewhat emaciated man. On the day of admission, pulse 102, range of temperature 2·2°—from 101·2° to 103·4°. No albuminuria.

March 23rd.—The pains in the ankles have subsided; there is no swelling or tenderness about any of the joints here or elsewhere, but there is considerable tenderness with some slight redness over a limited space in front of the left tibia. The right leg bears the scars of incisions made for erysipelas; the largest are seen on the outer surface of the limb, two on the thigh, and several more over the fibula. Besides these there are numerous smaller cicatrices of a coppery tint about the inner side of the right knee and over the upper half of the left leg. The edge of the right tibia is unnaturally rough. There is no œdema anywhere. The chest is voluminous and over-resonant generally, but deficient in clearness of percussion-note and in force of respiration in the right uppermost interspace. The first heart-sound is coarse and frapping. Pulse 108; range of temperature 2·6°—from 101·6° to 104·2°.

24th.—Slept for two hours before midnight. From this time he became delirious, restless, and talkative, twitching the face, and attempting to get out of bed. Towards morning he calmed down in some measure, but the twitching and the restlessness never disappeared. Urine retained in bladder, and drawn off by catheter. In the evening he was again delirious. Range of temperature 2·4°—from 101·8° to 104·2°.

25th.—Slept all night; this morning he is drowsy, and still twitches the muscles of the mouth and eyelids. Tongue dry, brown, and glazed; cheeks hollow. The left knee is full of fluid, and both shins are red and tender. Pulse 120; range of temperature 4·4°—from 101·2° to 105·6°. Near midnight, when the thermometer stood at the maximum, he was put into a bath at 98°, lowered subsequently to 80°. During immersion

his own body-heat was reduced 1·8°—from 105·6° to 103·8°; after removal it sank to 99°, giving a total reduction of 6·6° at 1 a.m on the 26th. He then seemed calmer than before, but it was more like the calm of exhaustion than anything else, for he complained of great weakness after the bath, while the drowsiness and the muscular movements of the face continued as before. There are now numerous patches of erythema, tender to the touch, along the tibiæ and over the back of the left hand. Quinine was given at 6.40 and at 7 a.m. in doses of fifteen grains, but made no impression upon the temperature, which in half an hour rose from 104° to 104·4°.

27th.—At 1.30 a.m. the fæces were passed involuntarily. He now became noisy, sleepless, and delirious, in spite of repeated sedatives. Breathing at 9 a.m. rapid and shallow; râles abundant throughout the chest; face and lips cyanosed. Left knee still full and fluctuating. Patches of ecchymosis are seen over the right tibia. A small pustule has formed on the forehead, and the right eyelid is œdematous. Range of temperature 2·4°—from 102° to 104·4°.

28th.—No change noted except that a copious eruption of large pustules has appeared on the face and thighs, and that the breathing is shorter and the pulse weaker than ever. Died at 3 p.m. Range of temperature 1·3°—from 102·8° to 104·1°; the maximum occurred at the point of death.

In addition to occasional measures of treatment, the following remedies were regularly administered:—First, salicylate of soda; then, on the occurrence of head-symptoms, chloral, morphia, wine, brandy, ether, ammonia, and bromide of potassium; finally, quinine to the amount of five grains, and tincture of digitalis to that of ten minims, every four hours.

Autopsy (from the Report of Dr. Sydney Coupland).—There was no excess of fluid within the pericardium. A few small hæmorrhages were seen under the visceral layer on the posterior surface. All the chambers of the heart were filled with clots. The clot in the left auricle was adherent and laminated. The wall of the left ventricle was rather thicker than natural and somewhat pale, but not of the faded-leaf tint. The mitral valve was considerably thickened, and the chordæ tendineæ stiff and coarse. The other valves were healthy. The right lung was slightly adherent to the chest-wall and to the pericardium. The lung generally was emphysematous. In the anterior part of the upper lobe some small whitish patches were visible on the surface. These were found, on section, to be wedge-shaped; they were of a dirty white colour, granular, and somewhat friable, but not actually purulent. The left lung was non-adherent, emphysematous, and dotted superficially with points of subpleural extravasation. On the inner and anterior aspect of the lower lobe was a small cuneiform mass, similar in appearance to those described in the right lung, but more softened, and in part quite purulent; it was about as large as a pea, and surrounded by a zone of intense hyperæmia. In the upper lobe were two patches resembling those in the right lung. The liver was enlarged, smooth, and pale; it presented no abscesses, infarcts, or subserous petechiæ. Spleen normal; rather small. Kidneys slightly adherent to their capsules; a cyst on the surface of one; surfaces of both, when denuded, distinctly granular and uneven at the edges; substance pale and coarse-looking; cortex diminished in bulk. The vessels of the pia mater were gorged with blood, but nothing else of any moment was discovered

within the cranium. There was no disease of the bony skull. The knee-joints were incised, and in the left a large quantity of sero-purulent fluid was found intermixed with flakes of lymph, A smaller amount existed in the right knee. There was also some puriform fluid in the right ankle-joint. Incisions made over the swellings on the left tibia and index-finger of the left hand let out some pus, which was situated, in the first instance, beneath the periosteum, and in the second case within the sheath of the tendon. The surface of the right fibula, along which the scars were ranged as aforementioned, appeared to be healthy on dissection. The upper tibio-fibular joint contained pus. The head of the right femur was dislocated, and found to be normal.

Gentlemen,—I presume you are all familiar with the chief clinical features of pyæmia as they present themselves in the domain of surgery, where they are far better pronounced than ever they are in the range of medical experience. To the surgeon, you know, rigors are of essential value and significance in the diagnosis of pyæmia. Without rigors he would at once look upon such diagnosis with the gravest misgivings—perhaps he would set it aside altogether as unworthy of acceptance. You must, however, bear in mind that to the surgeon belongs the privilege of witnessing and watching the majority of cases from the onset: many of the cases, indeed, are his own handiwork—the outcome of his own operations. Now, if ever there be a rigor at any time, it is sure to occur at the very outbreak of the malady, and the surgeon is sure to see or to hear of it. If he does not, he has strong *primâ facie* grounds for disbelieving in the existence of pyæmia. It is otherwise with the physician. Again and again the cases that fall under his charge are at least of some short standing, and the rigors may have come and gone unrecorded and unremembered, or they may have never existed at all. Recollect this, and in medical cases never allow yourselves to be carried away by the foregone conclusion, that there is no pyæmia because there is no *history* of shivering. Recollect, moreover, that rigors are really far oftener absent in the sphere of medicine than in the sphere of surgery, although they are the rule in both departments alike.

The symptoms of pyæmia are, in brief, a near approximation to those of enteric fever, with a large, but varying intermixture of the hectic form. The main distinctive criteria are the following:—If the rigors and the perspirations which commonly signalize the onset are repeated throughout the career of the case in disorderly fashion, you may safely exclude enteric

fever, typhus, ague, and acute rheumatism. Moreover, in the two first-mentioned the fever-spots are pathognomonic and cannot be mistaken for the sudamina and pustules of pyæmia. Again, the pleuro-pneumonia which may often enough attack the sufferer from rheumatism, and attack him severely too, is seldom at once so severe, so widely diffused, and so rebellious, as in pyæmia. Even the appearances around the joints, which make rheumatism and pyæmia look so like each other, may, for the most part, be discriminated with care. In rheumatism the redness is faint by comparison, and fades away insensibly into the normal hue of the surrounding skin; in pyæmia it is vivid and crimson-coloured, sharp in its definition and jagged at the margins. Such, at least, is the teaching of my own experience. Again, in the majority of cases of pyæmia there is an earthy-yellow tinge of the complexion, which in many instances may deepen into genuine jaundice. In rheumatism true jaundice is altogether exceptional, although there may be a trace of the characteristic tint in the eye and in the urine. In the common forms of fever jaundice is so rare as to claim especial recognition whenever it chances to co-exist. Far, however, above and beyond all other criteria is the testimony of the thermometer. The free and fitful oscillations on the chart are unerring and all-sufficient guides to the diagnosis. Nevertheless, it would be well to pause awhile and to enlarge upon the differential diagnosis of pyæmia and acute rheumatism. No two diseases are so often confounded; indeed, at first sight the confusion is almost inevitable unless there is something in the circumstances of the case positively to foreshadow the coming of pyæmia. I have made the mistake more than once for a moment, or for a longer though limited period; it would be unpardonable to remain in uncertainty or in error to the last. A few months ago I lectured on cerebral rheumatism—a form or manifestation of rheumatic fever which is most nearly akin to pyæmia, and this for two reasons: it is always accompanied by head-symptoms, as its name denotes, and it is generally accompanied by a high degree of fever-heat, often running rapidly into hyperpyrexia. In that lecture I besought you to stand on your guard, and to look ahead for the tokens of forthcoming mischief. I warned you then in all cases of acute rheumatism, and I warn you now in all cases that bear the least resemblance to acute rheumatism, when associated with nervous derangement, never on any account to allow the temperature to go beyond 104°, or at most 105°, before you resort to the bath. I acted on this principle in the

present case; I assumed it virtually to be cerebral rheumatism, as I was bound in conscience to do while as yet there was the shadow of a chance that it might accord with my assumption. In so doing of course I interfere with the natural changes on the thermometer. I do not give the instrument fair play and free scope for the delineation of the disease if, the moment it stands at 104° or 105°, I cool down the body-heat in the bath and reduce it in the end to the normal average, or below that level. This is true as far as it goes; the bath defaces for awhile, but it does not destroy, the distinctive readings on the thermometer—if you read between the lines; in other words, if you look at the lesser elevations, which are untouched by the bath, and disregard the main peaks, which alone are lowered by it. In well-developed cerebral rheumatism with high fever, the temperature will seldom fall spontaneously more than a degree or so; indeed, when left to itself it is almost always on the rise. In pyæmia, on the other hand, it may fall many degrees, so that with the aid of the thermometer alone you will not usually remain long in the dark as to the nature of the case. Again, apart from the thermometer, there is one cardinal criterion which in a multitude of cases will decide the point incontestably. In cerebral rheumatism, at the outburst of a paroxysm, the joint-pains *usually* subside or disappear. In pyæmia, if they exist at all, they pursue their own course; they have nothing whatever to do with pyrexia, hyperpyrexia, or head-symptoms. Perhaps I ought to have availed myself of this criterion in W. S—, in whom there was no abatement of pain or swelling at the time; perhaps I ought not to have bathed him at all, but I thought it best to be on the safe side, and to give him the benefit of a desperate doubt. It is worthy of note, as contrasting with the results obtained in rheumatic fever, that the bath made no impression upon the nerve-symptoms in our case: the subsultus and the drowsiness were just the same at the close as at the commencement.

I have pronounced the testimony of the thermometer to be an unerring and all-sufficient guide to diagnosis. It is so when the oscillations are present, but it must be borne in mind that they may be exceptionally absent. Their absence, then, offers no decisive presumption against pyæmia, provided there be collateral evidence in its favour. In my own case they were fairly, but not fully, represented. It is well also to remember that the variations may vanish altogether in any case on the approach of death.

What are we to regard as the primordial source of infection

in W. S— ? Not assuredly the lesions in the lungs, nor again the puriform exudations into the joints; these are well-known and stereotyped changes of a secondary order, proper to pyæmia. Dr Bristowe has traced pyæmia to suppuration beneath the periosteum; and to this source, it may be said, must we be fain to trace it in our own case for want of a better. I confess, however, that I feel some misgivings on this score. It may be, after all, that the true fountain-head and origin of the evil has never been discovered, and that the abscesses under the periosteum are themselves secondary. When the primary mischief is small and inconspicuous, it may well lie lurking in some sequestered and unsuspected spot. It is impossible to explore the innermost recesses of the entire body and it is pre-eminently hard to establish a negative in a post-mortem examination.

As for the treatment of the case, it would be mere waste of time to speak of it. The disease ran its course unchecked, and no measure of mine availed to postpone the arrival of death one single hour.

I have two more matters to lay before you. 1. The spleen is reported to be small. This is an unusual occurrence in cases of blood-infection, but it may occur in pyæmia exactly as we meet with it from time to time in typhus and enteric fevers. 2. I have introduced into the notes a term which may seem to call for explanation and even apology. I have described the first heart-sound as "frapping." The word is a homely one, but it is most expressive of the thing signified; that is to say, it expresses well and better than any scientific term a sharp first sound and a preceding momentary murmur or semblance of murmur—taken together. Provisionally, however, I speak of the first sound itself as "frapping."

I have many times made passing allusions to that brief auscultatory sign—that dwarf-murmur or mock-murmur—which is best designated by the name "preface" or "prefix" to the systolic sound. It may be described as appearing under two principal varieties—(1) as an undeveloped presystolic murmur —the faint foreshadowing of its possible mature existence; (2) as an alternative presystolic murmur taking the place of the mature murmur for variable periods, those periods lengthening as the murmur falls away in force of expression during the downward progress of the disease. In this latter case it might well be called a degenerate presystolic murmur.

That the sign in question and the recognized murmur of mitral stenosis are essentially one and the same thing, you may

convince yourselves by a few simple manœuvres. Take a pure example of the model-murmur, and, after ascertaining its undeniable presence at the apex, shift the stethoscope inch by inch from that point; you will find that the murmur shortens in duration and lessens in intensity at every move you make in the direction of the base, until at last it merges in the merest prefix; or you may vary the experiment, and apply your ear to the left scapula; the result will, or may be, precisely the same. Again, listen to the præcordia of your patient as he lies on his back in bed, and you may hear the preface and nothing more. Raise him, and make him lean forward; you may then recall at once the pronounced murmur and the concomitant thrill, if the thrill had no existence before, for it may coexist with the preface alone in all its forms and phases. We are forced to the conclusion that the substance and the shadow, the full-grown murmur and its diminutive counterfeit, are alike in their essence, and differ only or mainly in degree. Even the undeveloped form, with few exceptions, implies thickening of the mitral valve. Such thickening, you know, lies at the root of stenosis, and may become true stenosis if life only last long enough, but there is no stenosis of necessity for the time being. The alternative form, of course, has the same meaning with the murmur it replaces. Summarily, then, the sign possesses a more or less definite value; it signifies, as a rule, thickening of the mitral valve, whether it amount to stenosis or not. In the heart of W. S— thickening of the valve and its chordæ tendineæ was well displayed, but there was no narrowing of the aperture.

I have one more apology to make in this connection. I am in the habit of using the term "compound murmur," meaning thereby an apex-murmur consisting of two parts, separated, it is true, by the intervening first sound, but for the sake of brevity in description regarded as merging in one murmur, provided always the murmur be well expressed on each side of the natural sound. If it be ill-expressed before that sound—if the preceding element be nothing more than a momentary preface—it would be an excess of refinement to make use of a term which would require constant reiteration. The truth is, that over and over again, in cases of old-standing insufficiency of the mitral valve, we meet with at least the trace of a presystolic tone, although it may be much obscured when regurgitation is in the ascendant.

I am bound, however, to give you a word of warning here. Many auscultators might maintain that the so-styled preface is

no preface at all, but only part and parcel of the systolic sound itself, reduplicated, resolved into its elements, delayed in its evolution, roughened at the start, or otherwise modified. At any rate, they might put this construction upon my first variety. Some such modification there may be at times in rare instances; the point is a fine one to decide by an appeal to the carotid-pulse, the entire sequence of sounds is so swiftly past and gone. I can only say that to my own ear both the varieties afore-described are in the main alike and undistinguishable; in other words, to my mind they are both in the main presystolic. Nevertheless, I am often compelled by the exigencies of language to define my meaning in terms characterizing the systolic sound. I may then speak of that sound as unnaturally changed in various ways, although on my own view the sound itself may only be sharper and clearer than natural, while the remaining characters assigned to it really belong to the period of the presystole.

In conclusion, if I am not mistaken, if I have not strangely misunderstood the teaching of the wards and deadhouse, it appears to me that any one who seeks to give a good account of the full murmur of mitral constriction should first deal with the fragmentary form so often heard when there is no constriction at all. Stenosis clearly is not the sole item in the reckoning. How far will simple thickening go in explanation of the phenomena?

In the undeveloped form, apart from stenosis, it is hard to believe that a few patches of interstitial change can determine the result by mere obstruction to the flow of blood within the cavity of the ventricle, for it is here usually that the swollen patches would be floating at the moment when the brief murmur is generated. Again, in the undeveloped form, apart from stenosis, it is hard to believe that the loose valve should vibrate in its own substance and return vibrations to the surrounding blood. A membrane whose delicacy, though damaged, is not as yet destroyed, would never vibrate at all unless put upon the stretch and fastened home to the ventricular wall in every direction. It is otherwise with the developed form of the murmur and lesion as they exist in true stenosis. In this case the stiffened valve may vibrate, and not improbably such vibration is largely concerned in the genesis of the murmur and the thrill, just as the rude collision of the coarse-grained cusps may be largely concerned in the exaggeration of the first sound. What we want, however, is some common mechanism which will fit all the facts, and bring into harmony all the

varieties of presystolic murmur, in full length or in miniature. For my own part, I offer no solution of the problem ; I only wish to record my conviction that there is a problem to be solved even yet.*

* It has been suggested that the preface or prefix may only be a systolic murmur developed during the period presumed to intervene between the beginning of the systole and the closure of the auriculo-ventricular valves. It may be so, but, if so, on a review of the foregoing remarks, the strange conclusion is forced upon us, that every so-called presystolic murmur must be in some measure systolic or, more exactly speaking, must rest upon a systolic murmur as its base. Moreover, on this assumption we shall have to recognise a new order of systolic murmurs differing from the normal type in having the accent on the last syllable instead of the first. Fortunately, *on practical grounds*, it is a matter of no moment what the true rhythm and mechanism of the preface may be. Apart from all hypothesis, the sign has always the same significance; it points to the same anatomical lesions and bears essentially the same relations to the pronounced presystolic murmur, whether in its own right it is entitled to the name presystolic or not. Either it *is* presystolic in the proper sense of the term, or it is the representative and potential base of a presystolic murmur. On any view, the foregoing remarks may stand as they are with due reservations, *if necessary*, on the score of nomenclature. It is my own belief that no such reservations are necessary. The apex-thrill confessedly accompanies the presystolic murmur, and *may* accompany the mere prefix. Surely this is strong evidence in favour of an indentity of rhythm in the two physical signs, and as the one is beyond all question presystolic so apparently must the other be—in the main.

LECTURE X

ON A CASE OF ULCERATIVE ENDOCARDITIS WITH EMBOLISM OF THE BRAIN

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Mary T—, æt. 48, was admitted under my care July 31st, 1877. I am almost entirely in the dark as to her antecedents. She was unable to tell her own tale coherently, and there was no one to make good the shortcomings of her life-history, of which nothtng is known except that she denied ever having suffered from acute rheumatism. Respecting her last illness, I am indebted to Mr Lendon for the following notes:

On the 24th of July the patient complained of pain in the region of the liver and of uneasiness after taking food. On the 28th Mr Lendon was called to seen her at home. She was then complaining of incessant retching and vomiting and of obscure pain felt everywhere, but especially in the stomach, and over the left side. Her skin was hot to the touch, her tongue furred, her pulse 120, and her respirations 30. Nothing definite was discovered on examination of the lungs; there was, however, a compound apex-murmur, systolic and presystolic, with a concomitant thrill. The abdomen was slightly tender on pressure, but in no degree tense; there was gurgling in the right iliac fossa, but no fever-spots existed anywhere; the bowels were relaxed and the motions black, but she had been taking iron; the area of the splenic dulness was decidedly increased, and there was some pain in this region; urine of specific gravity 1024, non-albuminous.

July 29th.—The vomiting has been controlled by bismuth and hydrocyanic acid. Pulse 120; respirations 30; temperature 106°. She has been shivering for some time. Wandered during the night, but is quite sensible now. Expectorates a good deal of frothy muco-pus. No fresh physical signs. Appears to be utterly prostrate.

31st.—Pulse 126; respirations 33; temperature 105·8°. Tongue dry and brownish in the centre; bowels still relaxed. Physical signs as before. Received into the Middlesex Hospital this day. On admission, she is described as a dark-complexioned, well-nourished woman, with a pulse of 136, a temperature of 105°, and a breathing-rate of 44. Face flushed, skin hot, tongue moist at the margins, with a foul, dark fur on the dorsum; lips and gums sordid; abdomen slightly swollen, tympanitic, painful in parts, and generally tender over the entire surface. The area of the splenic dulness is enlarged, but the spleen

itself cannot be felt below the ribs, nor is there any tenderness here. There is, however, tenderness in the right hypochondrium where the liver dulness falls as far as an inch beneath the ribs in the mammary line. Gurgling may be detected here and there, but it is not limited to the right ilio-cæcal fossa. There are no fever-spots. Coarse sonorous râles may be heard posteriorly, but the percussion-note is fairly good everywhere before and behind. Slight occasional cough, but no dilatation of the nostrils. Her whole appearance is one of profound apathy and indifference to her condition; she delivers her answers slowly and with much hesitation; they are not always rational. No motion, no vomiting. At 6.30 p.m. temperature 104·6°, and at midnight 99°; range for the day 6·8°. Ordered a draught containing nitro-hydrochloric acid, also quinine, ten grains, to be repeated in twenty minutes; whisky, four ounces daily.

August 1st.—Slept well, but perspired profusely throughout the night. This morning she only complains of deafness, which may have been due to the quinine. No action of the bowels. Urine of specific gravity, 1020, non-albuminous. At noon temperature 105·4°, the maximum for the day. She is shivering again, but not violently. She is stupid and will not swallow; she cannot speak so as to be understood; she will, however, make a feeble effort to protrude the tongue when told to do so. Cough on the increase, although it is most abortive and executed with much straining. A compound murmur, chiefly presystolic, may be heard at the heart's apex, accompanied by a thrill. Left face blank; the right lateral furrow deeply indented; tongue deflected to the left. Apparently she possesses slight if any power of movement in the left limbs. Sensibility much impaired over the entire left half of the body, including the trunk. The eyes deviate to the right side. The left cheek bulges during expiration. Ordered tincture of digitalis five minims, with tincture of aconite two minims every second hour. An ointment containing belladonna and iodide of potassium to be applied to the præcordia. At 4.30 p.m. she was sweating abundantly, and at 7 p.m. the temperature fell to its minimum, 98°, yielding for the day a range of 7·4°. At 9 p.m. she appeared to be more helpless than ever. She tries to speak when spoken to, and to put out the tongue when desired, but cannot accomplish either act.

2nd.—Passed a restless and rambling night. No change in her general aspect this morning. Anæsthesia almost absolute; left conjunctiva impassive to stimuli. Over the entire left half of the scalp, face, neck, chest, abdomen, and limbs, deep punctures may be made with a needle without exciting any evidence of sensation, although the wounds bleed readily. Reflex action, however, may be easily provoked by irritating the sole of the foot. The pupils are about normal in diameter, equal, and equally sensitive to light. The eyes still deviate to the right; she will move them leftwards as far as the median line, but no farther. The head also inclines to the right, and cannot be pushed across to the left without producing pain. She will answer questions rationally, and her utterance is fairly distinct, but she is constantly wandering wide of the mark in her speech, and dwelling upon matters foreign to her present circumstances. All her passages are involuntary. Often she beats the chest rapidly with her right hand over the præcordia, but does not appear to suffer from any pain or discomfort there. At 9.30 p.m. pulse 144, respirations 64, temperature 104·2°. She is now perspiring freely, and seems hot and flushed. During

sleep both eyes are incompletely shut, the unclosed interval, however, is about the same on the two sides. At 11 p.m. the thermometer reached its maximum point, 104·6°, the minimum for the day being 98·4°; range 6·2°. Ordered solution of iodine to the shaven scalp.

3rd.—Temperature 103·4°, pulse 96, respirations 52. Lies in a state of profound sopor. At 12 noon she was almost comatose, breathing heavily At 1 p.m. temperature 105·8°. The compound murmur is now chiefly systolic at the apex, where the heart's impulse and the first sound are both strongly expressed. At 3.30 p.m. coarse laryngeal râles might be heard at some distance from the chest; the breathing was more embarrassed, and at times the patient appeared to be almost choked. At 4.30 p.m. temperature 105°, pulse 150, respirations 60. At 5.30 p.m. temperature 105·4°, pulse incalculable, respirations 60, shallow, irregular, often coming to a stand-still, interrupted by a fit of choking. At 7 p.m. temperature 106·3°. At midnight it stood at 106·9°, when the pulse marked 160 and the respirations 80. Maximum temperature 106·9°, minimum 101·6°, range 5·3°. Two leeches applied to the right temple; digitalis and aconite omitted.

4th.—At 1 a.m. temperature 107°; at 9 a.m. 105°. Wholly unable to swallow; nutritive enemata rejected; coma continues. At 1 p.m. temperature in axilla 107·2°; at 3 p.m. in rectum 109°. She was then dusky and pulseless. At 3.15 p.m. she died, with a temperature in the rectum amounting to 110·4°. Range for the day 4·7°.

After death, at 4.15 p.m., temperature in rectum 109·2°; at 5.15 p.m. 107·2°.

Autopsy. (Abridged from the report of Dr. Coupland.)—Head:—There was excess of fluid within the sub-arachnoid cavity. The vessels of the pia mater were full of blood. Lymph was found exuded on the inferior aspect of the cerebellum and at the base of the brain, especially around the chiasma and in the interpeduncular space. The right middle cerebral artery was swollen and plugged from its origin onwards, so that it felt like a piece of cord. From this some black clot extended into the right anterior cerebral artery, and into the terminal branches of the middle cerebral trunk. The territory supplied by the plugged vessel was completely softened, so that in raising the brain the organ fell asunder between the optic thalamus and the corpus striatum. The precise limits of the softened mass were not ascertained until vertical sections were made after hardening in spirit. The softening was then found to involve the whole of the corpus striatum in both its nuclei, with the exception of the lamina presenting in the ventricle. It was found also to involve the island of Reil and the anterior part of the optic thalamus. Moreover, the morbid change spread far into the circumjacent white matter and convolutions, but nowhere was it noticed above the level of the corpus callosum. Posteriorly, only the investment of the optic thalamus was softened, and not the body itself. There were no inflammatory lesions of any moment around the seat of softening, which was gray and curdy. Thorax:—Pericardium natural. Heart flaccid. Both sides, but chiefly the right, full of fluid blood and soft clot. Pulmonary and tricuspid valves normal. Endocardium of left auricle much thickened. Projecting into the auricle a polypoid mass of vegetations sprang from the margins of the mitral valve. Similar excrescences of smaller size occupied the posterior wall of the auricle. All the vegetations were loose and fragile, and in the neighbourhood of one of these a small ulcerated spot was discovered on the

posterior cusp. The chordæ tendineæ were greatly thickened, and the apex of the papillary muscle of the septum—the largest of the musculi papilliares—was converted into fibrous tissue. The mitral orifice barely admitted the tips of two fingers. Wall of left ventricle mottled and friable. Both lungs highly congested. At their posterior bases they were slate-blue coloured and collapsed. Abdomen :—The spleen measured five inches and a half by three inches and a half. It was pale and soft, and in its upper part presented a small wedge-shaped mass of a yellow colour surrounded by a zone of hæmorrhage. Liver large, dry, and glistening on section; lobulation ill-defined; consistency elastic, not friable. Cortical substance of kidneys swollen and pale, contrasting markedly with the medullary portion, which was congested. Surfaces slightly uneven and soft. No embolus in either organ. The uterus was retroverted.

Gentlemen,—Ulcerative endocarditis is a disease always characterized by enormous overgrowth of valvular vegetations. It has no necessary connexion with coexisting rheumatism, although, as a rule, the heart has suffered already from long-standing unsoundness. Its consequences are embolism or blood-infection, or both together. It is always accompanied by severe constitutional symptoms, usually described as those of intense pyrexia, but often possessing characters of peculiar type, and differing in some respects from the symptoms of violent fever in general. I have said the disease *may* involve blood-infection. In one sense it is always infective, for when the entire blood-stream flows bodily over an ulcerated surface, it can hardly fail to imbibe some taint of corruption, and to develop some degree of the fever of septicæmia. In the higher sense of the term it is not always infective; in other words, it does not always give rise to acute septicæmia, or to gangrene, suppuration, or other form of dangerous and degraded secondary change, for these alone are the proper products of infection, as I should understand it. I am bound, however, to warn you that by many authorities ulcerative and infective endocarditis are regarded as commensurate and synonymous terms. Nor is this all. Pathologists have brought to light the so-called material of infection, the presumed blood-poison itself, in the several shapes of micrococci, vibriones, bacteria, leptothrix-filaments and the like—a motley assemblage of molecular organisms which may be briefly designated microzymes. Clustered around the valves, or imbedded within the coagula of the heart, these infinitesimally small beings drift away in the blood-current along with the detritus of the clot or the vegetation, and either found colonies in emboli of their own

making, or else roam at large, in perpetual motion: in either case spoiling the purity of the wholesome blood. This state of things bears the name of "mycosis endocardii." Unfortunately no one can tell where these strange beings are born, whether *within* the heart or *behind* the heart in the course of the circulation, whether they are indigenous or exotic, central or peripheral in origin. Cases are on record which one would unhesitatingly refer to an extraneous source. On the other hand, there are undoubted examples where no distant point of departure was *discovered*, but in this connexion it must be borne in mind that the same thing is true of unequivocal pyæmia, in which over and over again we find no aboriginal focus of infection, whether anything of the kind exist or not. In all my own recent cases of ulcerative endocarditis post-mortem examination revealed nothing to warrant the assumption of an extraneous source. You cannot fail to see the importance of the question at issue in this matter. Can the heart spontaneously become the hotbed of a living mortal poison, or must it wait until the carriers of the poison are engendered elsewhere, and thence conveyed to the centre in the mature or in the embryonic form through the medium of a morbid lesion, or from first to last by way of the natural pores and passages of the body? The question, you see, most materially concerns the doctrine of abiogenesis, or the generation of life from no-life. In one respect the issue is unimportant. Whether or not the heart be in any sense the native soil of these mysterious molecules, at all events it is said to be a nursery ground wherein they grow, increase, and multiply at a fearful rate. In my own cases I cannot be sure that they had any existence at all during life, although in one they were discovered after death.

Let us concede that mycosis endocardii may perchance be present universally—not indeed in every instance of ulcerative endocarditis, but in every instance of the infective class as before defined. Another question remains to be asked. May we not learn a lesson from mycosis endocardii in the pathology of pyæmia? May we not see our way more clearly to the solution of the great problem, how to account for those secondary changes which, seated in front of the heart within the domain of the systemic arteries, are yet derived from sources of origin behind the heart, in the path of the venous circulation? Some instances of ulcerative endocarditis are themselves derived from this origin—in a word, they are examples of true pyæmia. In such cases there is no mystery

at all. Starting from the point of invasion at the periphery, and then marching severally in single file, either alone or in company with molecules of clot, microzymes might indeed pass the barrier of the lungs; but if they were to continue on their way severally, or even in groups of a few cohering corpuscles, they would never at once block an artery or even an arteriole, whatever mischief they might do to the capillaries. On the other hand, let us suppose that the heart is their halting place, and that here they rest for awhile and reinforce their numbers; they may then start anew, and, along with materials gathered from the heart itself, may advance in masses amply competent to stop the highway in the arteries and set up embolism there. As a rule, however, in pyæmia there is no ulcerative endocarditis, and therefore no mycosis endocardii, as the term is usually understood. Nevertheless, there may be analogous agencies at work, practically if not literally amounting to mycosis. The living organisms, and even the non-living portions of the blood-poison, when they arrive at the heart, may be free to enter into new arrangements there under new conditions. In lieu of the even onward flow of the venous circulation we have now to deal with eddying streams or stagnating pools. In these the septic elements may come into collision one with another; they may coalesce and cohere; they may react physically or chemically upon the normal constituents of the blood, so as to develop masses where before there were molecules only. With the right side of the heart we have no concern: moreover it is easy enough without these agencies to account for the products of pyæmia in the lungs. The left heart is all-important, and here the blood is aerated, another circumstance which may have some bearing on the result. All this may appear to be wild and fanciful in detailed description, but if the secondary changes beyond the heart in pyæmia are really owing to embolism of the systemic arteries, there is much to be said for the main idea. Surely in this case the material for impaction must be elaborated in the heart, or its elaboration must begin there. If, on the other hand, the secondary changes are not owing to the cause assigned, we can only refer them in the last resort to simple thrombosis at the seat of change, or, better still, to embolism of the capillaries properly so-called. Limited to those capillaries in the beginning, the block will extend slowly backwards by overgrowth of accessory thrombi, or small superadded plugs, until the entire mass of sequestered tissue assumes the site and the proportions of a true arterial infarct. Let the patho-

logists of the future determine all these questions—at present they are involved in the deepest darkness.

It is otherwise, I rejoice to say, in the clinical discrimination of ulcerative endocarditis, which becomes, for the most part, clear as daylight when the teaching of a single case points the way to the diagnosis of another. You have only to do your bounden duty, and, as a matter of course, to examine the heart of every human being that comes under your care, and it is well-nigh impossible to make a mistake. Such examination, however, is absolutely essential, for this reason. Vegetations may be growing in rank luxuriance over the valves, and yet, strange to say, there may be no præcordial distress, no local *symptom* of any sort to awaken the suspicion of mischief at the heart. Put your trust, then, in the physical signs, and, in association with the general symptoms, they will proclaim the presence of the disease with no uncertain sound. They may not, indeed, tell the whole truth at once; but wait awhile, and after one or two examinations the question will be set at rest all but indisputably. The physical signs are evermore changing from day to day in correspondence with unceasing changes in the form, site, and volume of the vegetations. The systemic phenomena are severe pyrexia, and, if infection be in the ascendant, then, in full force, all the symptoms of septicæmia.

I have said that the fever of ulcerative endocarditis is often in some respects peculiar and *sui generis*. I am warranted in so saying on the assurance of two of my three last cases. In one the temperature was never noted as exceeding 102·4°, whereas the pulse ran high throughout, and in the end inordinately high. In another, that of the girl H. C—, whom you must all remember, on a few occasions the pulse marked 150; many times it ranged between 130 and 140, and never once declined below 100 from admission to death—a period amounting to three months. The thermometer, on the other hand, never showed a higher reading than 103·8°, the minimum being 97·4°, the pulse at the same time registering 138. Surely this is a strange disproportion, and one that may possibly shed some light upon the diagnosis of the disease. Both the foregoing cases were of long duration, and in both the determining cause of death was hyperæmia and œdema of the lungs, exactly as might have happened at any moment to anyone suffering from heart disease without the aid of a blood-poison to accelerate the issue. The case of M. T—, differed widely from its predecessors. She died within a fortnight, and in her the blood-poison reached a high pitch of

intensity. Moreover, in her there was no disproportion of the kind above-mentioned, or if it existed at all, the ratio was reversed, and the temperature transcended the pulse-rate. Perhaps the hyperpyrexia, which closed the scene, may be reasonably referred to acute brain-softening, with meningitis, the consequence of that softening, and not to simple septicæmia. Be that as it may, it is the hyperpyrexia characteristic of severe shock or injury to the nervous system at its climax, and finds its nearest and best parallel in cerebral rheumatism, and occasionally in tetanus and lesions of the spinal cord. In M. T—, however, the temperature, though fitful and oscillating as in pyæmia, still maintained a high general average all along, and often reached or passed the uttermost limits of pyrexia proper, even before the occurrence of embolism in the Sylvian artery. Are we, then, at liberty to say that in the milder forms of ulcerative endocarditis the pulse-rate is ruled by the bulk and burden of the vegetations, provoking the heart to excessive action, but not materially exalting the fever-heat; while in cases that are truly and thoroughly infective the blood-poison is the overruling power that governs the course of events, and gives full expression to the fever in all its phases?

Other symptoms there are, or there may be, to which I should accord a high value and a strong significance. First let me mention a marked apathy in the patient, an utter indifference to everything and everybody around him, sometimes alternating with delirium. Whenever I have found a murmur or a medley of murmurs at the præcordia, I have learnt to look upon this frame of mind in the light of a symptom suggesting embolism of the extreme arterioles of the brain, chiefly in the cortical system of the convexity. M. T— exhibited unconcern and indifference enough, apparently owing to sheer helplessness and abolition of all mental power. Usually I have seen it in the guise of reverie and dreamy abstraction. Such demeanour was not to be expected in M. T—. In her the Sylvian artery was plugged at the main, there was extensive softening of the central ganglia and their environs, and there was meningitis. In the girl H. C— dreaminess was a conspicuous feature, and in her brain we found a minute focus of yellow softening near one of the parietal convolutions.

Another special symptom, and a most inauspicious one when it occurs, is an eruption of spots resembling petechiæ, and evidently originating in capillary embolism. As for embolism on the large scale, endangering life or limb, even when its

presence is fully ascertained, I exclude it from the reckoning altogether, and for a good reason. I wish you to understand the case with all possible speed before such embolism arrives, and not to await its arrival.

Last, but not least, in the catalogue of special symptoms is profuse drenching perspiration, second only, if it be second, to that of ague, and far beyond the average mark and measure of phthisis or abscess. In all my recent cases of ulcerative endocarditis I have invariably met with this superabundant sweating, so that, in my mind, it is now fairly enrolled among the leading indicia that would guide me on the way to a right diagnosis. Sweating, however, though generally symptomatic of the worst cases, is not universally present in all.

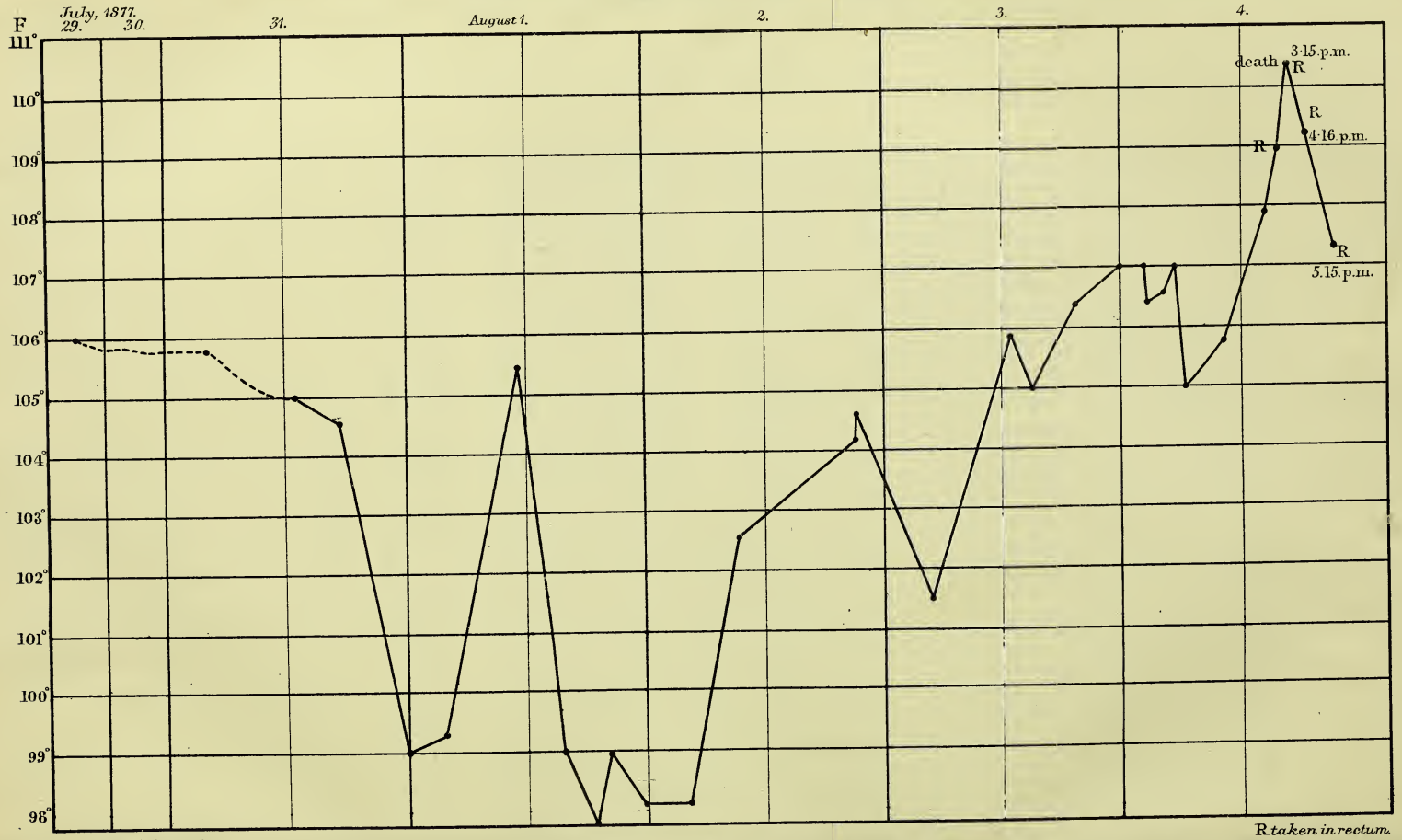
Finally, I have to comment upon the insensibility of the entire left side, associated with left hemiplegia, as a result of the embolus in the opposite side of the brain. Hemianæsthesia, intense in degree and bounded with geometrical precision by the median line of the body, is far from uncommon as a symptom of hysteria, if we may accept the accounts of the French authorities. Briquet and Charcot assure us that hemianæsthesia, perfect in all its points, occurs in 93 out of every 400 cases—that is to say, in upwards of 23 per cent. of all hysterical women. I can hardly imagine it to be so common in England; indeed, I am sure it is not; but then we are far behind the French in all the mysteries and marvels of hysteria. In the vast majority of instances you will be at no loss to discern the underlying neurosis, which will ordinarily be certain to betray itself by one or more of its familiar features, although you would do well to remember that M. Charcot has met with mixed cases truly cerebral and truly hysterical at once. Nay more, he has encountered a few cases of purely cerebral origin presenting all or nearly all the characters of hysterical anæsthesia. With reference to the pathology of the cerebral form, M. Charcot, after long wavering between many regions whose morbid changes might possibly determine hemianæsthesia, comes at last to the conclusion that the posterior part of the internal capsule is alone responsible for the result. At any rate, it is the only part which is found to be invariably destroyed or damaged in all cases of predominant and persistent hemianæsthesia where the lesions are circumscribed. Now, the internal capsule lies wholly within the domain of the Sylvian artery, a domain in the present case utterly disorganized by interception of its blood-supply at the fountain-head: hence the hemianæsthesia. The hemiplegia needs no

commentary. Cases of this kind are not numerous. The reason appears to be that the plug is usually seated at the point of a bifurcation, and not at the origin of the vessel. It is interesting to note how closely the lesions follow the lines of anatomy. The softened structures are all comprised rigorously within the range of the Sylvian artery; the unsoftened portions of the great central ganglia lie more or less beyond that range; their blood comes wholly or partially from other sources—in the corpus striatum from the anterior cerebral artery, in the optic thalamus from the posterior cerebral.

Regional diagnosis of brain disease offers many beautiful problems for solution. Has it any corresponding advantages in practice? It is a sad tale to tell, but I am afraid those advantages have been immensely overrated. For all that, I should be the last person to disparage or discourage it in its proper place, chiefly as a matter of medical science. On general principles, I should say in medicine, as in everything else, let us by all means solve all the problems we can. We never know what fruit our investigations may bear in time to come, however barren and unpromising they may seem to be now. Again, in a few particular instances, localization may enable us to forecast the future of a case. If, for example, there are grounds for ascribing the symptoms to the medulla or its environment, then beyond a doubt life is most seriously compromised. Darker still are the prospects of recovery when we have reason to believe that an extravasation of blood has forced its way freely into the ventricles of the brain. Finally, if we ever meet with another case of hemiplegia and hemi-anæsthesia conjoined, and know that it is the result of embolism, the chances are that the embolus must be lodged *in limine* at the very commencement of the Sylvian artery, over and against the points where the basal branches are given off from the main trunk. I need not tell you that a lesion so situated would be imminently perilous to life. If modern views of the vascular anatomy of the brain may be trusted, it is hard to believe that there exists in the arterial system around the spot any mechanism competent to save from utter destruction the ganglionic territory within the sphere of the Sylvian artery, however it may fare with the convolutions and the cortex. Not that I should refer the rapidity of the softening process to this cause alone. Large allowance must be made for collateral agencies, for the fever of septicæmia, and for the general depravity of the blood and tissues. Even in our case the constitutional symptoms and the nature of the lesion must

be taken into account. The mere site of the mischief in a particular region of the brain is only one item in the reckoning, and in the majority of cases it is only a subordinate item. Lastly, let me ask, will regional diagnosis avail you much in the matter of treatment? I fear not. It availed me nothing, or next to nothing, in my own case. I had only or chiefly to deal with the pyrexia and the prostration. What in the world could I do with the embolus? As for the meningitis, I knew nothing of it before death, and, if I had known of its existence during life, no measure of mine would have made the slightest impression upon it.

CHART OF TEMPERATURE.— Mary T.



LECTURE XI

ON A CASE OF PERICARDITIS WITH NUMEROUS COMPLICATIONS

Delivered March 21st, 1879. Published in 'Medical Times and Gazette,' November 15th, 1879.

Sarah B—, a married woman, æt. 46, was admitted into the Middlesex Hospital, January 3rd, 1879. Her father and mother lived to a good old age, the one dying at seventy-five the other at seventy-four. Her brothers and sisters are all healthy. Her own history is thus briefly recorded:—She has been subject occasionally to slight rheumatism in the finger-joints. She has never been laid up for any length of time. For the last twelve months, however, she has been losing flesh and becoming pale. About two months ago she was seized with shortness of breath and cough, which have grown gradually worse of late, but she has never suffered from pain of any moment. A fortnight ago œdema appeared in the lower extremities.

On admission she is described as a stout, flabby, anæmic, muddy-complexioned woman. Her legs are swollen as far as the knees, but there is no swelling elsewhere. She now complains of pain across the chest. She is short-breathed and coughs slightly. The lung-resonance is imperfect over the left front, excessive in the central regions at the back, but again deficient at each posterior apex. The heart's impulse cannot be felt, and the sounds are feeble and distant. The area of præcordial dulness reaches the second left interspace. The cough is paroxysmal; the sputa are thin and watery; the jugulars large and distended—markedly so in the act of coughing, when they become beaded and varicose. Urine of specific gravity 1015, non-albuminous.

January 5th.—A faint impulse may be felt over the fifth interspace. Over the third space there is a distinct systolic superficial murmur.

7th.—The heart-sounds are inaudible at the normal situation of the apex, scarcely audible in the fourth space, and very weak in the third. There is no murmur now, but the rhythm of the natural sounds has a slightly cantering character.

9th.—The right front is freely resonant, except over the breadth of an inch along the margin of the sternum. On the left side there is absolute dulness in the third, fourth, and fifth spaces, while in the second, and even in the axilla as far as its posterior border, the resonance is greatly impaired. Urine 1010, non-albuminous.

18th.—There is dulness in the axillary regions on both sides, reaching as high as the fourth space. Numerous fine moist râles throughout the lower lobes. No albumen in the urine; specific gravity 1020.

21st.—On the left side there is no good resonance anywhere in the

front or in the flank, except at the outer part of the two first interspaces. The axilla is dull even to its summit.

24th.—A distinct percussion-wave may be transmitted across the abdomen.

28th.—Grazing sounds heard superficially with systole and diastole.

February 6th.—A friction-murmur at the præcordia.

On the 7th heart-sounds unnaturally rough and murmur-like. Rhythm cantering. On the 8th the hands and arms swelled slightly. On the 11th she died.

I have above limited myself almost exclusively to a mere sketch of the main physical signs, drawn in the most meagre fashion. For the rest, the history was one of increasing œdema, ascites, dyspnœa, and distress, in no respect differing from the melancholy records of heart-disease in general. The respirations ruled high, and so did the pulse-rate. There was nothing noteworthy in the temperature. For a few days after admission there appeared to be some slight amelioration in the symptoms, but it was short-lived only. Soon the disease ran its course unchecked and uninfluenced by remedies, which proved to be palliative at best, and nothing more. It would be wearisome and useless to recount those remedies; let them pass.

Autopsy (from the report of Dr. Sidney Coupland).—Both lungs universally adherent to the chest-wall, the diaphragm, and the pericardium. The pericardial sac occupied a large space; its outer fibrous coat, overladen with fat, was much thickened and toughened in its proper substance. The visceral membrane was similarly thickened in a minor degree. Externally there were no adhesions between the pericardium and the chest-wall. Internally the heart adhered rather firmly to the back of the pericardium by a layer of recent lymph presenting a rough, honey-combed, granular appearance. When denuded of this material the heart was seen to be largely overgrown with fat. The apex was rounded, the right ventricle—distended with blood-clot—alone entering into its formation. The wall of this ventricle was mainly composed of fat, within which the myocardium was reduced to a lamina of soft friable tissue of a brownish faded-leaf tint. The wall of the left ventricle was similar in colour to that of the right, but softer in consistency, and more easily broken down. Microscopic examination showed that the muscular fibres were finely granular, but the striæ were not entirely obliterated. The valves on each side were nearly natural. The lining membrane of the aorta was raised into irregular prominences of atheromic deposit. Both lungs presented general condensation of their substance; they were tough everywhere, and the lower lobes particularly condensed and semi-carnified. A quantity of serous fluid flowed from the smooth dark-red surface on section. The liver displayed a typical nutmeg character. The spleen was soft and adherent to the left extremity of the liver. The capsule of the spleen was the seat of a dense yellow semi-cartilaginous patch. The kidneys were dark red, full of blood, finely granular on the surface, and greatly indurated.

Gentlemen,—The nature of the foregoing case during life was looked upon as a problem awaiting its solution in the dead-house. Of course it was a case of pericarditis; there could be no question about that point. Pericarditis, however, failed

utterly to cover the whole assemblage of signs and symptoms, not to mention that there was no unequivocal explanation of the pericarditis itself. The wide area of dulness or impaired resonance was far beyond the mark of pericardial effusion, even when its range is inordinately enlarged. Once, and once only, have I known an exception to this statement; but I venture to say that for fifty years to come we shall never meet with its like again. In this unparalleled case nearly the entire front surface of the chest was dead-dull on percussion, and nearly the entire breadth of its cavity occupied by what looked like a leathern bag full of fluid, while the heart lay at the bottom of the sac bristling with lymph and much resembling a hedgehog or a porcupine on its lair. In the second place, dropsy, as far as I know, never arises from present pericarditis alone, or from pericarditis immediately past. Something more than this, something deeper-seated and of older standing, was required in order to account for all the phenomena. Many wild hypotheses were advanced, many dreams of diagnosis passed before us in swift succession, and were as swiftly discarded. One fixed idea took possession of my mind, and held it to the last. There must be a morbid substance gripping the heart and hampering its action—not simply dislodging the organ, as a mass of cancer in the mediastinum might do. Accordingly, I made bold to write down as my diagnosis, fibrous induration of the pericardium with adhesions to the surrounding pleuræ. The diagnosis, you see, is true as far as it goes, but it falls short of the whole truth; the heart's chambers were found to be dilated, and its walls degenerate and overladen with fat. The dilatation, however, I regard as a mere corollary from the original lesion, the fibrous enlargement of the pericardium. Anything which embarrasses the free play of the heart-walls sooner or later leads to dilatation of their cavities, to say nothing of the old visceral pericarditis which may have undermined the cohesion of the adjoining muscular tissue, and lessened its power of resistance to the blood-pressure. To this dilatation the dropsy was owing in the main. Mere development of fat, whether in the form of growth or in that of degeneration, whether extra-fibrous or intra-fibrous, will seldom or never determine dropsy in the proper sense of the term. On the other hand, the granular changes in the kidney, slight as they seemed to be after death, and undisclosed as they were by albuminuria during life, may have lent their aid in the determination of the dropsy. For the disclosure of these granular changes we are indebted to the post-

mortem inspection alone. One of the great mysteries of the case was the uniform absence of albuminuria. Had we discovered but once even a trace of albumen, pericarditis would have been perfectly intelligible; it is a common accompaniment of the atrophic form of renal disease, and the præcordia should always be examined for it in these cases. As it was, we had no historical clue to the presence of pericarditis, which should never arise spontaneously; it is always, I believe, secondary to some prior ailment which ought to declare itself by its own peculiar signs and symptoms. On the whole, the antecedents of the case tell us nothing, or next to nothing; but here the autopsy comes to our aid, and sheds a clear light on the genesis and development of the disease. Everything points to the granular kidney as the *fons et origo mali*, save only the overgrowth of fat on the heart, which in all likelihood was owing to general obesity. Given kidney-disease at the onset, the pervading pleurisy on the two sides of the chest is at once explained. Again, given pleurisy and its products around the mediastinum, all the remaining phenomena follow in due order of succession one upon another, or they flow collaterally from the common source at the fountain-head. Pericarditis—external and internal, old and recent—degeneration of muscular fibre, increasing diffusion of dulness due to increasing œdema and condensation of lung-tissue, may all be referred with ease and certainty to the self-same cause. In this connexion you may well wonder how it came to pass that a disease, even now immature and unannounced by albuminuria, could have wrought so much mischief long before admission at a time when you might fancy the morbid change to have been insignificant and inappreciable to the naked eye. I would not have you to count too surely on the correctness of this idea. For aught we know to the contrary the lesion may have existed in much the same degree of development for months—nay, for years past. I have good grounds for believing that nothing in the whole compass of pathology is so slow in the cycle of its changes as granular atrophic interstitial nephritis. Bristowe says it may last ten years or more; my own impression is that it may last from fifteen to twenty years. You are all familiar with the corresponding lesion in the liver, known by the name cirrhosis —a name, however, which I hope you will never apply to the kidney. The two diseases are the counterparts of each other, not in structure only and in outward appearances; they are just as closely allied in the slowness and secrecy of their growth. Many a man is at this day walking about the streets

of London with one or other of these maladies in embryo, but without the shadow of a suspicion that he is the victim of a dangerous disease. Slow, however, as they both are in their evolution, the pace of cirrhosis is far faster than the pace of renal degeneration, although cirrhosis is not of necessity mortal, which the kidney disease must be in its own due season if nothing intervene to forestall it. Again, in the same connexion, note, I pray you, the absence of albuminuria from beginning to end—for a period of five weeks or more. Gentlemen, if you have reason to suspect that the kidney is degenerate, whether you base your reasoning on the age and aspect of the patient, on the life that he has led, or on the nature of the case, and the coexistence of symptoms otherwise hard to understand, never abandon the presumption you have formed, simply because you find no albumen in the urine although you may have searched for it unceasingly day after day. Still less abandon that presumption if the specific gravity rule low—nay, abandon it not if the density be of the normal average or above it. A low density speaks forcibly in favour of a dwindling kidney, but it is far from being universally present in these cases; often enough it is absent when the organ has not made much progress in degeneration, or when the case is drawing near to its close, or when there are severe complications in the chest. Least of all abandon the idea in question because you fail to discover casts under the microscope. This is a piece of negative evidence altogether untrustworthy. Again and again you will see no vestige of a cast even when the urine is albuminous. Do not mistake me, however. Suspect the existence of Bright's disease as strongly as you may, and even act on the suspicion as far as you are personally concerned, but keep your own counsel; tell it not to the patient, publish it not to his family and friends, unless there be albumen in the urine.

I reserve to the last the main point I wish to dwell upon. It is noted that there were friction-sounds and friction-like murmurs from time to time at the præcordia, and the heart-sounds are more than once described as cantering in rhythm.

The rhythm of the auscultatory signs in pericarditis is peculiar and almost unique in a large proportion of cases, amounting, I should say, to more than one half, perhaps to three fourths, of the cases wherein those signs are widely diffused and fully pronounced. It is a cantering tripartite rhythm—a tune in three times—you might waltz to it very well.

I feel bound to lay before you my own views on this matter, although they are hopelessly at variance with those of two

distinguished writers—Dr Hyde Salter and Dr George Johnson. The rhythm is composed of three several sounds, so arranged that two of the number go together while the remaining one stands apart. The solitary element, the monad, comes first in order, and the dyad or the twin elements follow and close the circuit. Moreover, to my ear, the monad coincides with the carotid-pulse and is systolic; the dyad falls within the period of the diastole about the time due to the diastolic sound.*

In the simplest form of the physical sign, to the best of my belief, we have only to deal with the splitting of the natural heart-sounds into three elements combined in the manner aforesaid: the systolic sound remains single, the diastolic sound is doubled. But what becomes of the adventitious sounds, the brushing murmurs, the rasping, the grating, and the creaking noises of pericarditis? Do they comply with the law that governs the evolution of the natural sounds in the simple form? To a great extent they do. The softer varieties, those which are more or less murmur-like in tone, in a multitude of cases conform to the law exactly. According to circumstances the natural sounds may or may not be abolished by the supervening murmur, but the rule is that the rhythm remains or comes into existence if it never existed before. It is then associated with the sounds and with the murmur, if both are heard simultaneously; with the murmur alone if the sounds have disappeared and the murmur alone is audible. In other words, the murmur itself is tripartite. It is otherwise, or it may be otherwise, with the harsher modifications, those which offer no analogy to a murmur at all. So soon as the signs of pericarditis assume the rasping, creaking, or grating characters, the voice of the rhythm is apt to be drowned in the din, and often vanishes altogether.

I dare not deny that there may be other varieties of tripartite rhythm in pericarditis owning a different origin, and standing in other relations to the carotid-pulse, but they are rare by comparison, and the standard variety is that which I have endeavoured to define. Again, I dare not affirm that the rhythm, as defined, is absolutely pathognomonic; it may perchance occur in some most exceptional cases—few and far between indeed—apart from pericarditis. Still less would I limit the *cantering* rhythm to pericarditis alone, although that is the most convenient term for common use. Triple peri-

* Dr Salter and Dr Johnson both make the elements of the dyad auricular-systolic and ventricular-systolic; the monad they represent as diastolic.

carditic rhythm would be a long, unwieldy phrase; besides, it would seem to involve a diagnosis—which the language of clinical medicine should never do, if it can be avoided; it should be descriptive only, wherever it is feasible. Bear in mind, however, that the two terms are not coextensive in meaning; they are thus far synonymous, and no farther. Every rhythm of the foregoing pericardial type is cantering, but every cantering rhythm is not typically pericardial. Cantering, then, is the larger term in the latitude of its range: it includes the other, and it includes more than the other. Still, in speaking of pericarditis, I should recommend you to adopt the expression, and, whenever you have a chance, to master thoroughly the thing expressed.

I have described the rhythm when it appears in its simplest form and is reduced to its lowest terms as a morbid subdivision of the natural sounds, essentially unconnected with the friction-signs of pericarditis. This point might be disputed, but I have repeatedly demonstrated examples of the kind wherein there was nothing to convey the remotest idea of a rub as ordinarily appreciated by the ear. In such cases it is utterly impossible that the natural sounds should be lost to the listener, or even much subdued in tone; they must be present and fairly well pronounced. In a word, they are apparently the first and second members of the trio. What, then, is the third member? If the first and second are not of the nature of friction-sounds there is no reason in the world why the third should be supposed to be of this nature. Possibly by making pressure on the chest with the stethoscope we may sometimes call forth a true rub, but this is of no importance to the main issue. What were the sounds heard before the pressure was made, when the rhythm revealed no degree of resemblance to the murmurs or noises of friction? The pressure-test may occasionally determine the origin of a doubtful murmur; but here you have no murmur at all to deal with. It might indeed be alleged that an amount and kind of friction incapable of altering the intonation of the two natural sounds may at the same time develop in their train a third sound—a friction-sound pure and simple, though devoid of all friction-like characters. This is fine steering, gentlemen; in my opinion, far too fine to be safe and sure.

I hold to my own conviction, that all the members of the entire trio in the simple form are only the natural sounds under a thin disguise; the third is but the echo and double of its predecessor. If this be the rule for the simple form, the same

will be the rule *mutatis mutandis* for the great majority of cases associated with genuine friction. In other words, whatever determines the triple rhythm of the natural sounds determines also the triple rhythm of the friction-sounds. What does determine the rhythm? It would appear that there is something which delays the evolution of the ventricular systole on one side of the heart in comparison with the other. Hence a corresponding delay in the diastole and in its accompaniments, the closure of the semilunar valve and the recoil of the distended artery. Now, pericarditis will paralyse more or less the underlying muscles of the heart-wall just as peritonitis paralyses the muscular system of the intestines. Indeed, the muscles themselves may be engaged in the inflammation. If, then, we are at liberty to suppose that pericarditis in the ordinary run of cases will not always or even often attack impartially the entire membrane, but may involve one part in a higher degree than another, we have at once the conditions of disharmony in the play of the ventricles. Where the exudation of lymph is slight, or where there is no exudation at all, there will be a simple splitting of the diastolic sound, a resolution of the natural sound into its separate elements. On the other hand, when the lymph is more or less freely exuded, without being so gross in bulk, or so coarse in grain, as to annihilate all rhythm, then there will be a double diastolic rub, owing to unsymmetrical expansion of the two ventricles. In many cases both sets of phenomena will concur at the same time in the same person. Moreover, the two sets of phenomena may interchange places in the same person at different times, in accordance with the changing phases of the circulation. When the heart is acting feebly, the simple rhythm may take the place of the rhythm with the rub, and the rhythm with the rub again take the place of the simple rhythm when the heart's action is strengthened.

It may be asked why, on the above hypothesis, there is no doubling of the systolic sound or the systolic ventricular rub in the characteristic rhythm? The reason is plain. In one respect the ventricles will harmonize in their movements; they will *begin* to contract at the same time, and no amount of disharmony in the strength or in the duration of the systole will ever double the sound or double the rub. It is otherwise with the diastole, which is delayed from the start on one side. Hence the diastolic sound and the diastolic rub will originate at different times on the two sides, and make a double impression upon the ear of the auscultator.

I lay no stress on all these speculations, but I find nothing in the disclosures of the dead-house to bar their adoption. On the contrary, post-mortem appearances go far to support them in cases of average severity. With the severest cases, and with the largest and coarsest exudations, we are not concerned. In these, as before said, the rhythm may be annulled altogether.

Let these things go for what they are worth. I do not ask you, gentlemen, to accept one word of theory or analysis that I have uttered this afternoon. I do not even ask you to recognize critically and musically the grouping of the several sounds described, so long as you recognize the rhythm itself in a practical way—so long as you know it when you hear it. Whatever you may think of the minutiæ involved in this discussion, the rhythm itself is no immaterial refinement. It is a matter of great pith and moment in the diagnosis of a disease which surely ought never to pass unnoticed. It is of the more moment, inasmuch as the simple rhythm alone, apart from all semblance of rub, is far from uncommon in pericarditis, especially at the onset. If, then, you pay no heed to this note of warning when it is sounded in your ears, the disease will steal a march upon you, and you will be so much the later in overtaking it with your remedies.*

* Dr Stokes, in speaking of the signs and symptoms of coming pericarditis, mentions among the rest as not uncommon, a doubling of one of the natural sounds, *most frequently the second* ('Diseases of the Heart and Aorta,' p. 93). Clearly, then, Stokes recognized the simple rhythm as a note of warning at the onset of pericarditis. The simple form, however, *may* sometimes persist throughout the whole career of a mild case. Again, Stokes would appear to have recognized the rhythm with the rub, but in this form he failed to see a *characteristic* feature of pericarditis. (Cf. 'Opus citatum,' pp. 29, 30, 76.)

LECTURE XII

ON A CASE OF MULTIPLE ANEURYSMS IN THE HEART, THE LUNGS, AND CAVITY OF THE CRANIUM

Delivered July 7th, 1876. Published in 'Medical Times and Gazette,' July 21st, 1877.

HENRY T—, æt 20, a draper's assistant, came under my care, in Cambridge Ward, January 12th, 1876, with the following history, as recorded in the notes. The only serious illness he can recollect arose from the kick of a horse over the lower part of the chest, on the left side, between eight and nine years ago. Within half an hour of the accident he spat blood, but never afterwards. On this occasion he was confined to the house for six weeks. His present attack began about a month ago, with chest-pains and shortness of breath.

On admission.—The cheeks were flushed purple; the general appearance was that of debility and emaciation. Complained chiefly of pain at the heart and across the loins. There was a loud blowing murmur of extreme intensity—loudest, indeed, over the upper and central regions of the præcordia, but loud everywhere in front, where it effaced the first sound altogether; and, again, well pronounced, although subdued by comparison, at the back. Coincidently with this murmur, a purring vibration or thrill could be felt at the base of the heart and over the whole of the right ventricle, but not beyond the boundary of the sternum; nor, again, leftwards, so far as the outermost limits of the apex-beat. The second sound was unnaturally rough at the base, and both sounds obscurely expressed in the carotids. The murmur distinctly and entirely preceded the pulse at the wrist; it was synchronous with the pulse in the carotids, or in part antecedent to it. At the posterior base of the left lung the first sound could be heard, apparently prefaced by a brief tone or bruit. The superficial veins of the neck were enlarged, and beaded in places, but they presented no pulsation.

As the case progressed, the boy began to look gaunt, haggard, and extremely ill. He spat blood; he suffered for many days from pain in the upper part of the right chest; and, when that abated, from pain at the left posterior base, where the percussion-note was dull, the breathing tubular, and the voice-sound bronchophonic. All the while the original pain at the heart continued, and all the while he was losing flesh and strength. The urine contained a trace of albumen, with an admixture of blood, as shown by guaiacum.

Such is a summary of the case as far as February 11th. From that date to February 17th the symptoms were chiefly those of dyspepsia—nausea, vomiting, and loss of appetite. On the 17th he coughed a good

deal, again spat blood, and suffered again from sharp pain near the right axilla. Temperature 104·4°—the highest on record. For many succeeding nights he required injections of morphia to procure sleep. The nausea, the vomiting, and the anorexia continued with interruptions; and it was not until the beginning of March that the pain subsided.

March 16th.—A double murmur may be heard at mid-sternum; both sounds muffled, and murmur-like in carotids. Pulse jarring. Pain, aggravated by deep inspiration and by coughing, below left mamma.

April 8th.—He has been wandering in the night, and even now seems confused. He speaks slowly, and with much thickness of utterance. His vocabulary is limited; for the most part he repeats a monotonous sound, which at times is clearly "Have mercy," and with this expression, plainly or indistinctly pronounced, he closes every sentence. The power of grasping is about the same in both hands, nor is there any want of symmetry in the lines of the forehead or face. The tongue, however, swerves in some measure to the right side.

9th.—Repeated the Lord's Prayer after the chaplain fairly well. On being asked how he was, he replied, with some hesitation, "I am first-rate." He cannot read audibly, however, nor could he repeat on dictation "A fine clear morning." Most of his sentences still relapse into the old "Have mercy." Wrote his Christian name, "Henry," quite well and legibly. When desired to write "morning" he failed altogether, but succeeded in copying a few letters of the word, presenting it under the form "mony."

20th.—Still there remain traces of asphasia, although, on the whole, he expresses himself with greater freedom and with a wider range of language.

27th.—Looks more and more ghastly, wan, and wasted.

May 18th.—Seems in great distress, and complains of pain and tenderness in the left side of abdomen.

29th.—The physical signs have undergone a change, The original murmur is much softened, and the diastolic sound is clear everywhere over the chest and in the carotids. There is now pulsation in the cervical veins, and yesterday there was slight œdema in the right foot.

June 1st.—After a long interval of clear, intelligible speech, he again miscalls many things. Looks deadly pale, and seems indifferent to life. Asked for a knife to cut his throat. From this time forth he sank, and died June 3rd.

Autopsy (abridged from the report of Dr. Sidney Coupland).—Beneath the arachnoid a large amount of fluid overspread the shrunken brain, the membrane itself here and there presenting patches of opacity with small plates of bony material. The left temporo-sphenoidal lobe, superficially orange-coloured, showed on section a cyst occupying the anterior half and containing a fluid like curds-and-whey. Seated on the middle cerebral artery, about half an inch from its origin, a saccular aneurysm, the size of a chestnut, blocked the entrance into the left Sylvian fissure. Internally, the aneurysm was found to consist of a compact mass of fibrine arranged concentrically in a laminated fashion, except in the innermost parts, where it was channelled and filled with soft black clot. The vessel was empty of blood. The heart weighed fourteen ounces and a half. On the right side the conus arteriosus was immensely enlarged; its walls were abruptly and inordinately thickened, and its cavity contained a mass of fleshy fragile vegetations, springing from the dense, irregular, milk-white endocardium, and

spreading as far as the attached margins of the pulmonary valve. Half an inch below the attachment of the valve, the septum ventriculorum in its anterior part was the seat of two perforations—one circular, just admitting a crow-quill, the other triangular, measuring three eighths of an inch in each direction. Both apertures were fringed with warty vegetations, but, in front and below, the margins of both were round, smooth, and continuous with the condensed endocardium of the conus. The foramen ovale was closed, and there were no traces of the ductus arteriosus. Projecting into the wide cavity of the left auricle, a group of vegetations arose from the adjoining borders of the two mitral curtains, close to the septum; and above, over the space of about a square inch, the opaque thickened endocardium was covered by vegetations. In laying open the left ventricle, a pouch was cut into, lined by a thick membrane, continuous with the endocardium through a smooth narrow orifice which occupied the interval between two diverging columnæ carneæ. The pouch was seated near the base of the heart, partly in the ventricular walls, but principally in the septum. The parietal portion was small; the septal division was oval-shaped, and measured an inch and a quarter in its long diameter from above downwards; its depth was half an inch; its smooth lining membrane was traversed by numerous folds marking the course of the muscular fasciculi which enclosed it on every side; it passed right through the septum, and opened into the conus arteriosus by the two foramina before described. Recent vegetations sprang from the ventricular surface of the aortic valve, but nowhere involved the free border. All the valves except the tricuspid were competent to the water-test. The right lung was firmly adherent at the apex. Two cavities of large size and long standing occupied respectively the upper and middle lobes. With these exceptions, the parenchyma of the lung presented no peculiarities of any moment. There were, however, five saccular pouches—true aneurysms—discovered on different branches of the pulmonary artery, all possessing walls of immense thickness, and all loosely attached to the tissues around. One was seated on the main artery supplying the middle lobe; it was of the size of a Barcelona nut, and lined by a layer of adherent fibrine. Four were found in the lower lobe, two as large as the last described, and two of smaller dimensions. They were nearly all engrafted on points of bifurcation in the artery, and, like the vessels, they were all empty of blood. No thrombi were found anywhere. On the left side some recent lymph coated the lower lobe, which was in great measure solidified, showing numerous tracts of embolic pneumonia varying in size and appearance, some entire, some broken down into irregular cavities. Large firm fibrinous clots were seen to block the branches of the pulmonary artery in this lobe, and in one of these branches an aneurysm was situated—the only aneurysm discovered in the substance of the left lung. In the upper lobe near the apex, was a recent infarction, of the size of a walnut, with a plug in the artery leading to it. The central third of the spleen was entirely composed of a large, wedge-shaped, buff-coloured mass, just beginning to soften in places. The remaining organs presented nothing that need be described.

Gentlemen,—Many a time and oft have I pointed attention to the foregoing case as a marvel and a mystery past all precise

interpretation. During the lifetime of the patient I could only waver between two alternative hypotheses—congenital malformation with an unclosed interventricular septum; and aneurysm in some unwonted form or place. Strange to say, although the hypotheses in question together covered the main mischief in the heart, yet neither the one nor the other proved to be absolutely correct. In giving the diagnosis I never dreamt of aneurysm perforating the septum; still less could I foresee the discovery of eight disseminated aneurysms. There can be no manner of dispute as to the nature of the phenomena; the wonder is how in the world they came into existence. As for the aneurysm in the septum, possibly it may owe its original development to the kick of the horse that occurred eight or nine years ago. Injury will cause laceration of the heart-fibres, and then, as you would naturally expect, it is the right ventricle which bears the brunt of the damage. Under these circumstances, let the heart-fibres be once torn, contused, or weakened on the right side of the septum, they will offer less resistance to the preponderating blood-pressure from the left heart. The blood bearing upon the intercolumnar spaces of the left surface will tell forcibly on the unresisting fibres of the septum; it will work its way into the substance of the partition-wall, and there excavate a chamber for itself, in close contiguity to the damaged structures on the right side. Given all these conditions, it is easy to imagine that the blood may break through the barrier altogether, and unite the two ventricles into one. Possibly the right ventricle may co-operate with the left, and excavations commenced at the same level on the two surfaces of the septum may in due course of time meet midway in its substance. On this view, or on any hypothesis implying time spent in development, the aneurysm in our own case, slowly tunnelling and at last perforating the septum, may have given origin to endocarditis, with its rank and exuberant overgrowth of vegetations. On another view the chain of causation may have been reversed, and endocarditis may have originated aneurysm in the acute form by a swifter process of disorganization. The seat of the aneurysm might seem to bespeak an acute origin, but its characters conform expressly to the chronic type. A smooth, narrow mouth in the groove between two diverging fleshy columns, an expanded sac, a dense lining membrane continuous with the endocardium at the orifice, and plainly showing on the walls of the cavity the mouldings of the muscular fasciculi, which appeared to maintain their integrity unbroken, and simply to have given way before the

advancing aneurysm; all these characters taken together present the very picture of an old formation. Moreover, the vegetations underneath the aortic valve, though, of course, near the orifice of the sac, nowhere came into immediate contact with it—a circumstance almost, if not altogether, irreconcilable with the idea of acute ulceration of the myocardium beginning on the left side, where we commonly find the point of departure for the ulcerative process. In spite, then, of all plausible presumptions to the contrary, the intraseptal aneurysm must have been an old one, whether we derive the endocarditis from its rupture or not. As for the remaining aneurysms—the six that were discovered in the lungs, and the solitary one that existed on the brain-surface—we may fairly ascribe their genesis to embolism. *Primâ facie*, indeed, if we were to look at the lung alone, we might demur to this opinion. The right lung, with its manifold aneurysms, offered no trace of plugging, infarction, or consolidation anywhere; the left lung, with its single aneurysm, displayed all these pathological changes on the amplest scale. Still, the drawbacks to the hypothesis are not insuperable. Emboli may give rise to aneurysms, exalting as they do the tension of the blood-pressure, and impairing the integrity of the vascular walls in their vicinity; and on a survey of all the circumstances the conclusion is irresistible that they have done so in the present case. Bear in mind the extreme rarity of aneurysm in one so young, the absence of any preceding flaw in the arterial system, the wide dissemination of the aneurysmal masses, coupled with the presence of a material at the heart, on the right side and on the left, ripe and ready for detachment and deposition anywhere at any moment—above all bear in mind the preference shown for the bifurcations of the blood-vessels, the chosen sites of emboli—and you cannot fail to see in embolism the sole sufficient cause of all the phenomena under discussion. Gentlemen, you recollect well the mysterious murmur described in the notes. Are we in a position to interpret this murmur now, with the heart lying before us? I fear not. So multifarious are the sources of murmur that I find it is impossible to recognize severally the part played by each in the production of the universal discord. Those who are familiar with malformations of the heart and its appendages may speak with authority on this point,—I dare not; I only venture, with many misgivings, to accord the foremost place to the pulmonary valve and the perforating aneurysm. In this connexion two circumstances deserve to be mentioned.

There were undoubted evidences of aortic valvular incompetency, alike in the characteristic pulse and in the characteristic murmur. Now, both these characters vanished towards the close of life, about the time when the boy began to complain of pain in the left side of the abdomen. What is the inevitable inference? A considerable cluster of soft and fragile vegetations were found attached to the under surface of the aortic valve, which, however, was pronounced to be competent after death on the assurance of the water-test. The inference is this. There must have been a time when the valve put forth a more luxuriant growth of vegetations, large enough to encroach upon its free margins and to interfere with the due play of its mechanism. Soon the encroaching material lost its hold on the surface, drifted away in the current of the circulation, and became impacted in the spleen, thus restoring to the valve the competency it had destroyed. The overgrowth disappeared, the undergrowth remained. The second circumstance is the following:—The long loud murmur in part preceded the systole of the ventricle: this point was determined by comparison with the carotid-pulse in front, where the first sound was inaudible over the chest. At the back it was ascertained by simple reference to the first sound, which was plainly distinguished at the left posterior base, and there only. The vegetations on the mitral valve and orifice, and on the adjacent surface of the auricle, possibly explain the presystolic element in the murmur. Possibly, also, the blood streaming through the aneurysm may have developed sonorous vibrations there, even during the period of the auricular systole. I have spoken of the carotid-pulse as a measure of time in the interpretation of a mumur; and this leads me to the lesson the case before us is pre-eminently calculated to teach. On applying the stethoscope to the præcordia with one hand, and placing the fingers of the other over the carotid, you at once recognized the rhythm of the murmur as systolic in the main. Again, when you laid your finger on the radial artery, the murmur seemed to fall within the diastolic or presystolic period. It concurred for the most part with the carotid-pulse; it distinctly and entirely preceded the pulse at the wrist. You were enabled to verify this by determining the close coincidence of the apex-beat with the carotid-pulse, in comparison with its marked priority in point of time to the radial beat. The radial pulse, you know, is a little later normally than the heart's impulse; but here the difference in time was abnormally great. You see, then, that in estimating the rhythm of any given murmur

it is unsafe to rely on the pulse at the wrist. Whenever it comes to a fine point, and absolute precision is required, you should as a rule, place your finger on the carotid while you are listening at the heart. Unfortunately, this is not always an easy matter to accomplish, at least with the rigid stethoscope in common use. The attitude is awkward and embarrassing, the carotid itself is but feebly felt in many persons, and often the slightest movement of the neck may utterly derange the best concerted manœuvres. If, then, you are foiled in your manipulation of the carotid, try the subclavian, just where it is on the point of dipping beneath the clavicle. The interval between the pulses in the two arteries must be immeasurably small. The subclavian is the larger vessel, and presents a pulse less liable to change and less easily effaced by the movements of the muscles, while the clavicle offers a rest that steadies the hand and leaves the ear and the head at full liberty to pursue their investigations in peace and quietness. Enough of the heart: let us pass to the brain. I need not remind you that in the vast majority of cases of aphasia, understood in its widest sense, the lesion is on the left side, and that in the majority of such cases the seat of the morbid change is the convolution of Broca or its environment. Pathology, then, and, I may now add, direct experiment, would seem to mark this region as a great physiological speech-centre for the mass of mankind; in other words, as one of the main links in the long chain of succession, from the supreme centres downwards to the nerve-nuclei that govern immediately the muscles engaged in the consummation of speech. Our own case is no exception to the rule in pathology. The Sylvian artery near its origin was blocked by an aneurysm, which lay imbedded on the surrounding structures, among the rest, on the convolution of Broca. The branches to the corpus striatum ran clear of the block; they arose in front of it on the proximal side. The remaining ramifications encircled the aneurysm, and must have been submitted to strong pressure. The temporo-sphenoidal branch appears to have been absolutely closed in some part of its course. The branches distributed to the gyrus of Broca and the island of Reil would seem to have collapsed and expanded in a desultory manner by intermissions. In accordance with the varying fulness and force of the cerebral circulation, or with the changing bulk, form, and structure of the aneurysm itself, they were more or less open to the reception of blood flowing at times in a slender stream, at times in volume sufficient to maintain the normal activity of

the speech-centre. Again, the pressure bearing upon the blood-vessels must have borne upon the brain-tissues around the aneurysm at the same time, in the same measure, and with the same variations. Hence it is easy to understand why the aphasia should appear, disappear, and finally reappear. I would willingly stop here, were it not that a distinguished professor—one of the foremost men of the day—by the mere ascendancy of his name has well-nigh thrown us back into chaos and confusion. Dr. Brown-Séquard denies to Broca's convolution and its surroundings all physiological power over the mechanism of speech. This doctrine is only part and parcel of a comprehensive scheme which aims at revolutionizing the pathology of paralysis, as hitherto accepted—a scheme, indeed, which goes far to render impossible all localization of brain-power. I am, however, only concerned with aphasia. Let us suppose, then, that our presumed speech-centre, now under controversy, is softened or otherwise damaged in its nutrition. You are called upon to believe that it is not the destruction of tissue, or the abolition of energy in the part diseased, that directly paralyses the apparatus of expression, but that the paralysis is owing to an irritation derived from the damaged spot, and conveyed away to nerve-cells in other regions, where it is empowered to put a veto on the operations essential to speech. The *modus agendi* is complex —first irritative, then inhibitory. Now, gentlemen, aphasia may last a man's lifetime without material change from beginning to end. Is it, then, conceivable that the processes described can go on for evermore without a break, and never let slip a single opportunity of silencing speech or circumscribing its scope? To my mind, unceasing, unchanging paralysis, the result of untiring excess or sustained perversion of energy in a nerve, is one of the hardest of all hard things to understand; harder by far than all the anomalies that beset the old belief in passive paralysis. Be that as it may—concede, if you please, all the profound and enduring influences claimed for irritation and inhibition, operating intermediately upon nerve-cells scattered broadcast throughout the brain—you are no nearer the mark practically. The region above defined is still in disease the main source of aphasia, whatever the nature and channel of communication may be. Even when robbed of all its authority over the utterances of health, it still remains a pathological centre—a centre of speechlessness, if not a centre of speech—and for all practical purposes we are exactly where we were.

LECTURE XIII

ON A CASE OF CARCINOMA OF THE PLEURA, LIVER, AND LEFT SUPRA-RENAL CAPSULE, WITH ASCITES

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W. D—, æt. 49, a remarkably large and powerful man, first a soldier and latterly a police-sergeant, was admitted on 30th of April last. The health of his family is good; his own antecedents had been exceedingly good up to November, 1872. His height was six feet, and his weight, when in condition, sixteen stone. He had suffered indeed from small-pox and scarlet fever when a child, and from fever and ague when on service in the Crimea, but during the whole period of his duty in the police force, comprising twenty-one years, he was never invalided for a single day. In November, 1872, while engaged in taking a prisoner into custody, he received a blow on his left side and was severely shaken; in a few days afterwards he began to spit blood in small amount, but never left off duty. At the same time he had most inordinate thirst, drinking several quarts of water during the night, and passing large quantities of light-coloured urine. For some weeks before admission he had been rapidly losing flesh and strength, and for a corresponding period he had suffered from severe pangs shooting down both legs from time to time, and from pain over the epigastrium and hypochondria and in the left lumbar region. He had always been a temperate man.

On admission.—Face bloated, eyelids puffy, conjunctivæ œdematous, cheeks mottled and streaked with capillary congestion. Free resonance over the whole front of the thorax; dulness at both posterior bases, where the breathing is weak; sonoro-sibilant râles everywhere. Area of heart's dulness apparently curtailed by overlapping lung; first sound at apex roughened; impulse in fifth interspace half an inch to inner side of nipple line, slightly undulating and diffused, occasionally intermittent. Abdomen voluminous, everywhere freely fluctuating; resonant in a high degree over a large space in the neighbourhood of the umbilicus, which is unfolded and flattened; dull in the flanks and along the pubes; dull also over the epigastrium and hypochondria, where the sense of displacement is readily elicited by strong and sudden pressure with the fingers. The lower margin of the liver can be distinctly felt reaching within two inches of the umbilicus, firm and rounded, in parts embossed. Legs highly œdematous; considerable œdema of scrotum and penis. Several spots resembling nævi over trunk and arms. Urine of specific gravity 1010, non-albuminous.

May 5th.—Less œdema; less distension of albumen; fluctuation now

limited to flanks. The breathing is exceedingly weak everywhere, and numerous fine crackling sounds are audible, especially at the right base, where these sounds present a near approach to the characters of friction.

From this date to May 10th there was considerable improvement: the abdomen continued to diminish, while the urine increased and became very abundant. During the whole period from the beginning he has been treated with hydragogues and diuretics, digitalis, squill, copaiva, and compound jalap powder.

May 11th.—Passed a good night, and looks more comfortable. At 9 p.m. he still presented the same appearance of comparative comfort. At 10 p.m., however, he seemed to be growing weaker, and to suffer from shortness of breath. At 11 p.m. the breathing became very laborious; loud bronchitic râles were audible, and distinct bronchial fremitus was felt extensively throughout the chest.

12th.—1.30 a.m.: Surface of body and extremities cold and clammy; pulse past counting. Died at 2.45 a.m.

Autopsy (from Dr Sidney Coupland's report).—The abdominal cavity contained nearly two pints of dark sanguinolent fluid; the intestines were much distended with flatus. The dura mater was unusually adherent to the brain along the middle line; the brain-substance appeared to be normal; the organ was very large, weighing sixty-one ounces and a half. The left pleural cavity contained eleven ounces of straw-coloured fluid; there were also one or two ounces on the right side, and a few ounces of serum in the pericardial sac. The heart was universally enlarged and hypertrophied—more markedly, however, in its left chambers; it weighed twenty-one ounces and three-quarters. The endocardium was somewhat opaque, and the musculi papillares in the left ventricle inordinately developed, almost creaking under the knife. There were some patches of atheroma on the mitral valve, the free border of which was irregularly thickened; all the valves were competent. The right lung was slightly compressed in its lower lobe. The right pulmonary pleura was covered by small opaque elevations, appearing under the microscope to be solely made up of fibrinous material, with corpuscular elements. Besides these, there were a few sparsely distributed nodules, averaging a pea in size, white and firm, limited to the pleura, and evidently cancerous in nature. The lung was nowhere freely crepitant; its tissue was tough, much congested, and œdematous, the anterior margins being emphysematous. The bronchial tubes were full of frothy secretions, and the lining membrane thickened, villous, and injected. On the left side the pulmonary pleura presented even more extensively the fine soft granulations above described, but only a small number of the cancerous nodules. The posterior two thirds of the upper lobe were solidified and in a state of gray hepatization; the rest of the lung was highly œdematous. The liver was immensely enlarged, weighing twelve pounds five ounces. Both lobes were equally infiltrated with cancerous nodules and masses in every variety of shape and size; one or two of the larger masses on the lower surface appeared to have been ruptured recently; each mass presented a white encephaloid appearance, and was surrounded by a zone of hæmorrhagic infiltration. Kidneys large, congested and indurated. Right supra-renal capsule healthy; left supra-renal capsule enlarged to nearly three times the size of the corresponding kidney, coarsely lobulated on the surface, and invested by a very vascular membrane. The entire capsule

measured six inches from above downwards, and four inches and three quarters in its broadest diameter from before backwards; it weighed thirty ounces. On section the mass could be distinctly separated into a small internal nucleus, and into an investing or cortical portion. The central substance was translucent in some parts, opaque and yellow in others, and ranged about two inches and three quarters in every direction. The cortical substance was soft and medullary in appearance, and in one part broken down. Spleen enlarged and softened; mucous membrane of stomach coarsely mammillated; intestines healthy; no notable enlargement of mesenteric or retro-peritoneal glands.

Gentlemen,—The man whose organs I have just laid before you, and whose case I have sketched, was one of the finest specimens of manly development it was ever my lot to witness. It would appear, also, that his constitutional strength must have been quite on a par with his enormous muscular power, when we take into account the many eventful years he passed in the Crimea, and his long service in the dangerous duties of a police-sergeant. I allude in passing to these points mainly by way of illustrating a well-known truth, that strength of muscle and hardihood of constitution are no safeguards whatever against the invasion of carcinoma, nor, indeed, afford the slightest presumption against the possibility of its occurrence. I have another motive in making this allusion. Immense muscular development, and a life spent in hard unceasing exercise, go a long way towards accounting for the extraordinary size of the heart. In no inconsiderable degree, however, I believe the heart is proportioned to the man; the volume of its muscular fibre and the capacity of its chambers in great measure only represent the magnitude of the muscular system at large, and the grand scale of the entire framework. After due allowance on this score, all the residue of real hypertrophy may be easily and naturally set down to the long-continued strain of a most laborious life. It is true that the *primâ facie* appearances of hypertrophy are most conspicuous in the left ventricle; there is nothing, however, very remarkable in this on any hypothesis, nor are they limited to that ventricle alone; if they were so absolutely, my explanation would fall to the ground, and we should be compelled to seek in a diseased kidney the first foundations of the hypertrophy. The kidneys, however, were not notably diseased; or, at any rate, they showed no traces of long-standing disease, which alone would account for the phenomenon to be explained. Let it, however, be distinctly understood that the heart here presented to you offers a fair approximation to the type of

heart you must expect to find in those who die of slow granular degeneration of the kidney. Let it be understood, also, that I am not denying all disease—or, at least, disorder—in the kidney. Apparently there was enough of the one or the other to account for the swelling of the eyelids and the œdema of the conjunctivæ, but assuredly not enough to explain the existence of a heart like this. Lastly, let us not ignore the special alterations discovered in the heart's structures, the opacity of the endocardium, and the thickening and atheroma of the mitral valve. It is only fair to take them into the reckoning, although they will not help us materially; they may have, indeed, impaired the natural purity of the first sound at the apex; but neither in themselves, nor in anything they imply of antecedent change, can they be regarded as standing in any close relation to the hypertrophy.

With respect to the several organs involved in the carcinoma, you may naturally ask me whether it is the liver, the supra-renal capsule, or the pleura that is entitled to priority of place as the starting-point in the train of mischief. Possibly it might strike you that the determining cause of the cancer may well have been the accident of November, 1872, the severe shaking, and in particular the blow received on the left side. Injuries and irritations have often been quoted among the recognized causes of carcinoma, and on this assumption you might pronounce in favour of the supra-renal capsule as the seat of primary contamination. Unfortunately, as far as I know, all modern authorities, with the sole exception of Rindfleisch, agree in stating that the supra-renal capsule is never attacked by carcinoma primarily; in other words, they have so far discovered no unequivocal example of carcinoma in this organ standing alone. You are bound, therefore, to set aside the supra-renal capsule, and inasmuch as the pleura for many reasons must come last in the series, you arrive negatively and by exclusion at the liver—the very organ which would at once positively present the strongest claim to the foremost place. Not that I would altogether ignore the influence of the blow: it may have been instrumental in determining the current of secondary contamination, so that it should set towards the supra-renal capsule in preference to any other organ. Strangely enough, the blow and the shaking seem to have been really instrumental in provoking a near resemblance to a brief and abortive attack of polyuria or diabetes insipidus, a disease known to have followed concussions or injuries. By way of parenthesis, it may be well to

remind you that bronze skin or Addison's disease has no connection with carcinoma, nor, indeed, with any other pathological change save the development of fibro-cellular tissue and its transformation into caseous matter. There was no trace of discoloured skin or mucous membrane in our patient.

We have discussed the origin of the malady. What determined the issue? It is singular that within the present year I have had to lecture on three cases of abdominal carcinoma, in none of which did the sufferer die the death of carcinoma properly speaking—death by inches, death by prolonged distress, by slow inanition, by exhaustion and emaciation. On the contrary, two of the number were at the onset in the possession of a fair amount of strength, and all died in the most rapid and unforeseen manner by acute engorgement of the lungs. One died in twenty-six hours from the invasion of the concluding chest-attack, and in him I ascribed the cause of death at the time in large measure to collateral pulmonary hyperæmia. W. D— died in five hours from the commencement of the final severe attack upon the lungs. Is it possible to ascribe his death in any appreciable measure to the same cause—collateral hyperæmia? I am of opinion that it must be so ascribed in some degree. The right lung was certainly curtailed in its proportions by encroachment on the part of the liver, and the respiratory apparatus on the left side was to some extent replaced by fluid in the pleural cavity. This, however, cannot possibly be all. The exclusion of pulmonary blood from one part, and its resulting accumulation in another, cannot, in W. D— at least, have been sufficient to account for death. Perhaps in all three examples considerable stress should be laid on another element of danger—the cancerous cachexia itself—the degradation of the blood and the wasting of the powers of life, which render the system vulnerable at all points and expose a man to jeopardy from attacks which would have been innocuous in ordinary circumstances. Of course I do not mean to say that fatal engorgement of the lungs is a thing to be positively apprehended in all cases of carcinoma in general, or in carcinoma of the abdominal organs: you must bear in mind that two of my patients were already suffering from chest-complaints, and under such conditions the possibility of danger arising from this source may fairly be entertained. On any view, it is a striking and suggestive coincidence that all these men should have died in the end of lung-disease; it arouses the suspicion that life in cancer, though protracted as a rule, really hangs upon a thread, and

and may be cut off at any moment. In this connexion, we may well wonder at the extreme tenacity of life displayed by carcinoma wherever it is suffered to hold the even tenor of its way without let or hindrance, as contrasted with the swiftness of its downward career under the influence of intercurrent disease in general—certainly under the influence of chest-disease.

I have one more point to discuss—the coexistence of fluctuation and free resonance over the same parts of the abdomen—a sign well exemplified in W. D—, and one which I have several times recently demonstrated to you at the bedside (I hope convincingly, as far as the fact is concerned). I will now endeavour to explain what I believe to be the mechanism of the fact, and in so doing I shall be obliged to enter into details; but I make no apology for this, inasmuch as, judging from the ideas ordinarily entertained by gentlemen in the wards, I should say that there must be something in the statements of the authorities on ascites, if not exactly erroneous, at all events obscure, unsatisfactory, and perhaps positively misleading. The general idea in vogue among students appears to be that there is a necessary antagonism between fluctuation and free resonance; that, in fact, they cannot coexist in the same parts. I trust I have shown practically that they can, and do often coexist. Let us examine the point *ab initio*. You are all familiar with the cylindrical contour of the intestines as they are described and figured in text-books and diagrams, and as they are seen bodily in dissections; and this you may, perhaps, regard as their natural shape. It may be so in some sense, but assuredly it is not their normal and physiological shape; it is not the shape in which they can by possibility exist in conformity with physical laws within the abdominal cavity of a living healthy man. In health, and even in disease, so long as there is neither fluid nor air within the peritoneal sac—nothing, in fact, to fill the interstices which must intervene between the adjoining convexities of two or more cylinders, given they are to maintain the cylindrical form—it is simply impossible for them to maintain that form. Consequently the walls of the intestine are flattened, collapsed, or otherwise deranged in symmetry by close apposition to each other, so that the area of a transverse section represents an irregular polygon and not a true circle. Now, let there be a somewhat scanty accumulation of fluid in the peritoneal cavity, and what is the consequence? You will have—in the ordinary recumbent posture—a region of percussion-dulness and fluctu-

ation more or less distinctly expressed in the flanks, and you will have a region of free resonance without fluctuation over the superior strata—those which are farthest removed from the plane of the bed, those which constitute, if I may so speak, the arch of the abdomen. So far there has been no material deviation from the physiological shape of the intestines; they are all floating above the water-level—folded and packed one upon another, much as in the natural state—the fluid is shallow enough and the mesentery is long and lax enough to allow of this arrangement. You see my terms are all throughout this discussion adapted to the small intestines; this is for the sake of simplicity; but remember the facts themselves are equally true of the large intestines, in so far as they are free to float, and not held immovably down by their respective mesocola. Now let the fluid go on accumulating until there shall be great but not extreme tension of the abdomen, and what is the consequence? The surfaces of the bowel are no longer under the physical necessity of accommodating themselves to each other by close apposition over extensive areas; throughout the peritoneal cavity there is "water, water, everywhere;" the intestines unfold, and assume the familiar cylindrical contour, and the water pervades and fills the crevices between the convolutions. Under these circumstances, you will have below, in the flanks, fluctuation with dulness absolute or comparative; above, in the central spaces around the umbilicus, or at any rate over the dome of the belly, wherever that may be, fluctuation and free resonance intimately combined and often widely diffused. In other words, you have in these superior regions both fluid and bowel beneath your finger, in close proximity to the wall of the abdomen. Either they are disposed in alternating succession exactly at the summit-level, if the mesentery at its full stretch be just long enough for this disposition; or, if the mesentery keep the bowel below the surface, the lamina of overlying fluid is so thin, and perhaps so easily dispersed by the finger-stroke, that practically the percussion-note remains unimpaired. Lastly, let the fluid still increase until the tension becomes immoderate. In this case, unless the mesentery be unnaturally long, the bowel is far removed from the surface by a thick overlying stratum of fluid; there is dulness and fluctuation everywhere over the abdomen; and if there be a trace of resonance anywhere, it will be under the walls of the thorax. Of the foregoing conditions the second is the most important; it is the commonest that falls under observation, for it implies just that degree of distension which compels a man to seek

medical advice. The condition below might pass unnoticed or unregarded; the condition beyond is almost incompatible with life, or at least unbearable. It is, however, to the physical signs of the second condition that I wish now particularly to bespeak your attention; I mean the concurrence of fluctuation and free resonance in the same parts, denoting the close contiguity of bowel and fluid to the very spot examined; the fluid in absolute contact with the wall of the abdomen, the bowel either equally in contact with it, or immediately below. I am not wasting your time in idle speculations. You may verify the point for yourselves at once on an emaciated living subject. The lightest stroke at the shortest distance will in the cases described transmit the wave across the resonant area from finger to finger. If you are not satisfied with this, go to the dead-house, and having previously ascertained on the body the existence of the associated signs in question, watch well the first incision made into the abdominal cavity. The instant the knife penetrates the peritoneum, there will be a rush of fluid from the sac, and a presentation of the intestine in its rounded form at the opening. Both are seen to be directly underlying the surface of the abdomen together. The wave is generated at the spot, and not propagated from below. Nay, you may have precisely the same proof given you in the lifetime of the patient if there chance to be oozing after paracentesis. I have more than once seen serum issuing from the wound when there was full and free resonance all around the very point of leakage.

Whatever the value of my explanation may be in its details, the main facts are assuredly true, and the corresponding physical signs are well worthy of attention for the following reasons.—In the first place, they prevent us from under-estimating the amount of fluid within the peritoneum. Secondly, in the presence of ascertained ovarian dropsy they lend us material aid in determining the question whether there is ascites superadded or not. Finally, in the vast majority of cases they render the diagnosis of ascites at once swift and sure, involving no inconvenience to physician or patient. It is but the work of a moment. Given the combination of signs aforesaid, you know instantaneously, as a rule with scarce an exception, that the fluid beneath your finger is in the peritoneal cavity, for there alone, as a rule, could the conditions exist that give rise to this combination. The drawbacks or sources of fallacy are few. You might by accident fix your finger on the border line between the intestines and an ovarian sac, and so

might elicit resonance from the one and fluctuation from the other. This might conceivably so happen, perhaps once in fifty times, perhaps once in a hundred examinations. It is, however, the easiest thing in the world to deal with this difficulty. You have only to enlarge the area of your manipulations by taking a circle of a few inches in diameter, and the fallacy vanishes altogether. A similar fallacy, to be dealt with in a similar way, might arise from the presence of a distended bladder overlapped at the margins by intestine. Again, it is just conceivable that an ovarian cyst might contain gas, the product of decomposition. Such things have occurred and might occur again, but how often? Perhaps once in three hundred examinations. Rare, however, as such contingencies are, still more rarely would they present the combination described. In ascites the fluid and the gas are contained, the one in the peritoneal sac, the other in the bowel, and under my second condition, of which alone I am speaking, both would be free to approach, and even to reach the summit-level collaterally and independently of each other. In the case supposed both are contained in the same receptacle, the ovarian cyst. Hence the gas would rise to the top and stand well above the surface of the fluid, so that the fallacy could only disconcert us at the precise plane of separation between the overlying and underlying strata. The same remarks apply to fæcal abscesses intermingled with air from the bowel, and to accumulation of fluid or semi-fluid matters above the ileo-cæcal valve in typhlitis, but here the percussion-wave alone would usually mark the difference; seldom, indeed, would it be so fine as the thrill of ascites. All these points may be settled at once by simple enlargement of the area examined. For all practical purposes, therefore, with the exercise of ordinary care and common sense, the test stands unimpeached, whenever and wherever we are able to realize its evidence. In W. D— the fluid rapidly subsided, and in a few days reached so low a level that the associated signs in question disappeared entirely from the uppermost parts and the experiment was useless.

LECTURE XIV

ON A CASE OF SYPHILITIC DISEASE OF THE LIVER, WITH PECULIAR PHYSICAL SIGNS IN THE ABDOMEN

Delivered March 12th, 1875. Published in the 'Lancet' August 28th, 1875.

W. H—, æt. 40, a hawker and a hard drinker, was admitted originally on the 17th February, 1874. He had always been a strong man, and for the most part able to do the work of a professional pedestrian —walking at the rate of six miles an hour for two hours together— until four years before the date of admission. He then suffered from pain in the left side of the abdomen, much increased by the ingestion of food. In the course of half a year he entered the Brompton Hospital, and remained under treatment there for nine months, with enlargement of the liver accompanied by severe pain. At the close of that period his liver, so he was told, had become smaller. He himself assigned six weeks as the duration of his present illness, and latterly, it is true, he had vomited from time to time, but his abdomen had always been large, and his pains had continued on and off in a slight degree, without increasing severity at any time, during the whole interval of three years and a half preceding admission. His disease, then, must have been of long standing, and his own reckoning must be set aside altogether.

On admission he presented the physical signs of ascites, along with a trace of jaundice in the complexion. Dulness over the liver region enormously extended in every direction, below over half the abdomen as far as the level of the umbilicus, and above as far as the second rib, but here the extension is obviously owing to an abundant effusion into the pleural cavity, occupying the lower two-thirds or four-fifths of the chest on the right side. The dulness, however, fails to represent the full measurement of the organ, whose inferior margin may be traced an inch further downwards by palpation than by percussion. Everywhere over the abdominal area of the liver-dulness a sense of displacement is experienced when the fingers are pressed sharply home on the subjacent mass, and everywhere the surface of the organ underneath appears to be broken into ridges and hollows. In the region of the spleen there is dulness reaching as far as the transverse umbilical line, and, on long inspiration, even below that level. The hand may be easily inserted into a narrow gap between the opposing margins of the liver and spleen. Heart's impulse undulating and widely diffused, yielding a very extensive thrill. First sound loud and clear, especially at the left apex, where it is slowly evolved, hesitates, and, as it were,

hangs fire. Ordered a powder consisting of tartarated iron and cream of tartar every six hours, and a draught containing copaiva three times daily.

February 19th.—Girth of abdomen rapidly diminishing, fluctuation subsiding; urine double the normal quantity.

24th.—Jaundice disappearing. Patient wandered slightly during the night, and in the morning vomited a porringerful of grayish-black, muco-puriform matter.

28th.—To the left of the umbilicus there is a delicate purring sensation or fremitus, plainly felt when the palm of the hand is laid lightly on the surface, but at once destroyed when the pressure is increased. At the same spot a humming sound may be heard on auscultation if the experiment be gently made, but the sound is at once abolished when the ear is applied firmly to the surface.

March 9th.—The fauces, and in particular the mucous membranes of the hard and soft palate, are intensely injected, and in many places pitted with hæmorrhagic erosions. Derived apparently from this source, large quantities of glairy-looking blood-stained sputa are expelled daily.

23rd.—The level of the percussion-dulnesss over the right back of the chest is now lower by three inches than on the date of admission, and all trace of fluid in the abdomen has disappeared. This day the patient received his discharge.

On the 18th of June, 1874, he was readmitted with signs of augmented effusion into the right pleural cavity. Thoracocentesis was proposed to him, but he resolutely refused to undergo the operation. He was again discharged on the 2nd of July.

On the 29th of December in the same year he was again readmitted in the following state:—There is dulness over the lower half of the chest on the right back, but free resonance from clavicle to nipple. No thrill can be felt at the heart's apex now, but the first sound is long, lagging, and briefly prefaced. The sputa continue to present the characters before described, and the membranes of the soft and hard palate are as vividly injected as ever. There are no physical signs of ascites. The left lower ribs below the mamma are much enlarged and rougher than natural, and in the same situation there is a prominent fluctuating mass, unaltered by position or pressure. The old purring and humming sensations may be elicited, as before, on the left of the umbilicus, but the seat of their production is no longer limited to that spot; they may be recognized just as clearly in the right hypochondrium under the same conditions.

On the 2nd of January, 1875, two ounces of laudable pus were withdrawn by aspiration from the abscess over the ribs. On the 26th occurred a sharp attack of fever, followed by commencing erysipelas on the face. At the same time a grayish-black, sloughing, superficial ulcer was seen to occupy a large portion of the velum and fauces. On the 31st hæmatemesis supervened on three several occasions, the blood lost amounting on the whole to about five or six pints. From this time forth to the close of life nothing of any moment ensued, with the exception of an attack of jaundice. The erysipelas, the hæmatemesis, and the ulceration of the fauces, all disappeared entirely, and the man simply sank and died on the 15th of February.

Autopsy (*condensed from Dr. Coupland's Report*).—Glands of groin enlarged; no scar on penis. Seated mainly beneath the periosteum of

the tenth rib on the left side were the remains of an abscess occupying the two adjacent interspaces, and containing inspissated cheesy material, in which was found imbedded a fragment of the rib lying bare, and detached from the rest of the bone. The left superficial epigastric vein was seen within the sheath of the rectus muscle to be greatly dilated; it was about the size of the little finger, and was traced towards the left and above the umbilicus, where it became very varicose; its further course was not followed, but a vein was observed on the ligamentum teres nearly the size of a crow-quill. The internal mammary veins were also enlarged and full of blood. *Thorax.*—On the right side the pleural sac was filled brimful with a clear dark yellow fluid, showing a sediment of paler colour and creamy puriform appearance at the back near the spinal column. The lung, collapsed and carnified throughout, was dragged away from the spine by bands of adhesion attached to the extremities, but allowing the organ to hang free at the sides in the mid-cavity of the pleura. The left lung was unadherent, non-crepitant, intensely engorged, and dripping with blood. The inferior margin of the lower lobe was compressed by the adjoining spleen and collapsed in its whole extent. The mitral valve was competent to a stream of water projected through the aortic orifice, but the cusps and the chordæ tendineæ were greatly thickened. *Abdomen.*—The peritoneal cavity contained barely a pint of bile-stained fluid. The liver was displaced downwards; bands of adhesion passed from its surface to the parietal peritoneum; they were most plentifully supplied with blood-vessels, which were fully injected, and all running from the liver to the walls of the abdomen. The liver itself was strangely misshapen, its lobes being broken up by deep furrows into a multitude of irregular knobs, while the whole surface was more or less granular. The capsule presented numerous thickenings unevenly distributed. There was also well-marked fibrous thickening at the bottom of the furrows and along the course of the larger portal canals, and here and there were seen small tracts of caseous character in the midst of the denser material. The liver-substance was throughout permeated and toughened by this fibrous change, so that it was nearly impossible to distinguish any individual lobules, the general appearance being that of fine granules, of various sizes and shades of colour, interspersed with larger masses of a dark green tint. The weight of the organ was 56½ oz. There was no dilatation of the interstitial bile-ducts; in fact, their presence could hardly be ascertained. The hepatic and common bile-ducts were also free. The spleen was immensely enlarged. It was adherent to the parietal peritoneum; the capsule was dense, opaque, and puckered; the parenchyma firm, fibrous, dark red, and liver-like. The mesenteric veins were fully engorged. The stomach was the seat of partial hour-glass contraction. The lining membrane was thick and firm, and everywhere of a deep slate-black colour. In the region of the fundus were numerous punctiform erosions, all surrounded by a zone of pigmentation, and some of their number filled with a black-coloured plug of altered blood. The entire mucous membrane of the intestines was exceedingly vascular and thick, while in the ileum the patches of Peyer were brought plainly into view on the red ground by their uniform slaty discoloration. The kidneys were enlarged, congested, and indurated. The testes were nearly normal. None of the viscera showed any trace of lardaceous degeneration. *Head.*—There was a clot found in the longitudinal sinus, and the vessels of the pia mater

were full of blood. The dura mater and the brain-substance were healthy.

Gentlemen,—On a review of the foregoing case, it is impossible not to recognize that the root of all the mischief lay in the inflammatory attack which occurred four years and a half ago. Inflammation, however, does not exclude syphilis; and, if we are to accept the pathology of the present day, to syphilis we must refer the strange disfigurement of the liver described in the notes, in spite of many and grave misgivings, on the score of the following antagonistic facts:—The man himself stoutly repudiated the charge of syphilis, although he freely confessed to gonorrhœa; there were no manifestations of the disease in question on the surface of the body; there were no unequivocal gum-like formations anywhere; above all there was no evidence of amyloid degeneration. On the other hand, the round-topped eminences, flanked on each side by a deep cleft, at the bottom of which runs a stream of fibrous tissue, for the most part along the channels of the larger portal passages—these are the very features which are held to be pre-eminently distinctive of the syphilitic liver in its outward configuration. They are, indeed, rough representations on the massive scale of what appears in the finer form and in miniature when the liver is simply cirrhosed. Not that cirrhosis is incompatible with syphilis; far from it, they are often associated. In our own case there was cirrhosis characteristically displayed in its truest colours.

Again, the caries of the rib and the abscess developed originally between the bone and the periosteum strike the balance in favour of syphilis, and far outweigh all presumptions to the contrary on the ground of the absence of gum-like formations or amyloid change. After all, the former may have had a real existence within the fibrous septa, and then disappeared, the caseous material remaining as the *débris* of their disintegration. In reference to the latter, it is, indeed, passing strange that syphilis should fracture a rib, and, while leaving its mark so strongly impressed upon the liver, should yet leave no trace of amyloid degeneration anywhere.

Clinically regarded, the following are the prominent points in the history of the case.—Under the influence of copaiva the ascites passed away, at least so far as to be no longer discoverable during life. Other remedies were administered at the same time, but to copaiva I am convinced must be awarded the priority of place in the accomplishment of the cure. I

have met with more than one marvellous illustration of the power of this medicine in dispersing ascites and restoring life where death appeared to be staring the sufferer in the face. It is, indeed, inconceivable that copaiva can in any degree modify the primary condition of the ascites—the disease in the liver itself. It is only a diuretic, but in many cases it is one of the surest and steadiest of its class, free and uniform in operation, allowing fair play to the overtasked veins, and giving them full leisure to recover tone and power to bear the strain of the exaggerated blood-pressure. In this way, by a course of strong diuretics aided, in some instances by paracentesis, in all by hydragogues and vapour baths from time to time, and by tonics throughout, we may indulge the hope of achieving a speedy cure of ascites in a minority of cases. In the majority the great point is to sustain the strength; the method of evacuation, though still indispensable, is now of less moment, and in the main we trust to the great physician Time, who works in a wholly different way, by the construction of new vessels or by the expansion of anastomoses already in existence, thus establishing what is known as the collateral circulation. In this view our own case becomes intensely interesting. I am not here called upon to enumerate all the by-paths in the circulation that conduct the portal blood to the right side of the heart when it is denied a free passage by its own high road through the liver. These are all well given by Dr. Goodeve in the article on "Cirrhosis" in 'Reynolds' System of Medicine,' and they were all beautifully illustrated by the disclosures of the dead-house above recorded. I would, however, lay particular stress on the series of new vessels that pervaded the false membrane, on the enlarged superficial epigastric vein within the sheath of the rectus muscle, and on the enlarged vein coursing along the ligamentum teres; whether with Rokitansky we look upon the last-named vessel as the unobliterated and engorged umbilical vein, or with Sappey we regard it as one of the accessory portal veins. Be that as it may, it is a matter of the deepest moment, in cases of cirrhosis, to ascertain, if possible, during life, whether the collateral circulation be fairly well established or not. What evidence, then, have we bearing on this point? Of course in a large proportion of cases we see the uppermost vessels conspicuously streaking the surface of the abdomen. Below the surface there runs an unseen auxillary circulation, whether conveyed by channels nominally superficial or nominally deep-seated. Have we any evidence of this drainage that is going on under-

ground and out of sight? With regard to the deeper strata of blood-vessels, I cannot say with absolute certainty that we have, but with reference to those which, though invisible, are yet immediately underneath the surface, our own case is conclusive as far as itself is concerned. We had the clearest evidence on this score in the purring sensation and in the humming sound perceived exactly over the spot corresponding to the enlarged superficial epigastric vein, and again towards the right hypochondrium over the seat of the vascular false membrane, and not far from the neighbourhood of the ligamentum teres. I dare not, however, presume to apportion precisely to the respective vessels their several shares in the production of the aforesaid phenomena, beyond simply saying that the superficial epigastric vein must have been their sole source of origin at the time when they were first discovered. Let that pass. The sign, if it can be verified by repetition hereafter, will possess some value with a view to forecasting the future of a case. I need not say that it will be a sign of good omen, for it signifies a free collateral circulation. This freedom of the circulation it will be our duty to develop and maintain—principally by tonics, in order to strengthen the reconstructive energies of the system; in a minor degree by the method of evacuation, in order to lessen the amount of fluid within the abdomen, and to relieve tension, which, over and above other evil agencies, would choke the blood-vessels and preclude their development. Well, we cured the ascites, and in all likelihood the collateral circulation prevented its recurrence in any degree appreciable during life. It did not, however, prevent the recurrence of that hæmorrhage into the stomach which was the main element in the multitude of causes that led to the man's death, and perhaps for this reason. In cirrhosis of the liver it is quite true that hæmorrhage into the alimentary canal is in great measure determined by mechanical congestion; but there are secondary processes at work. The blood or the blood-vessels, or both together, must have undergone degradation; for hæmorrhages occur in other regions far away from the portal system, not only in cirrhosis but in a variety of liver-diseases. This point also is amply illustrated in our case by the blood-stained sputa and the prolonged engorgement of the mucous membranes of the palate and fauces. In truth, the hæmorrhagic erosions of those membranes so conspicuous during life were the very counterpart of the appearances revealed after death in the membrane of the stomach. We thus arrive at the melancholy truth that in

removing ascites even for a permanency, we do not remove all the dangers of cirrhosis: the danger from hæmorrhage still remains.

I have not dwelt upon the use of iodide of potassium. Of course, in cases like the foregoing, it must be tried by way of a preventive measure in order to stop the ulterior progress of advancing syphilis. Beyond this it will be of no avail. When once the characteristic changes in the liver are well pronounced, when once ascites and hæmorrhage have supervened, iodide of potassium will no more undo the mischief that has been done already than it will abolish a cicatrix. I gave it a fair trial, and found it utterly useless.

LECTURE XV

ON TWO FATAL CASES OF ASCITES

Delivered March 3rd, 1878.

GENTLEMEN,—You have seen many a life saved or prolonged by paracentesis in ascites. Let me reverse the picture and show you some of the dangers you may incur by resorting to the operation.

John N—, æt. 42, a publisher's assistant, was admitted September 11th, 1877. The following is an abstract of the notes taken at the time. He has been an intemperate man and a dram-drinker. He suffered from dysentery in the Crimea, and has contracted gonorrhœa and syphilis twice in his life. For a considerable time he has had an occasional heavy pain in the right hypochondrium. Soon after its accession the contiguous chest region underneath the right inferior ribs appeared to bulge, and the ribs themselves to be unnaturally large. A fortnight ago the pains became paroxysmal, and he was obliged to retire from his occupation. He then noticed for the first time that his abdomen and lower extremities were beginning to swell. Three weeks before admission he passed blood by the rectum.

On admission he is described as a fairly well-nourished man, with a certain nervousness of manner, and a marked tremor of the tongue. The legs are tense and œdematous from the knees downwards; the skin over the insteps is shining and fissured, and the entire limb on each side studded with subcutaneous petechiæ or spots of purpura. The abdomen is volumnious and globular, measuring forty inches and a half at the umbilicus, and presenting all the characters that point to the existence of fluid in abundance within the peritoneal sac. The breath-sounds are feeble at both posterior bases. He complains of dyspnœa and thirst; the skin is harsh, and the tongue thinly coated and dry. Temperature 99·7°; pulse 100.

In the evening he was tapped, and on the following day it is noted that the abdomen, though diminished in size and offering a more extensive area of resonance, still remained conspicuously large, and that there was a decided increase in the œdema of the penis and scrotum, although the lower limbs were much reduced in bulk. Urine of specific gravity 1030; about four ounces passed in twelve hours; loaded with lithates, but yielding no albumen, and no blood on examination. On the 13th a trace of albumen was discovered. The conjunctivæ were slightly tinged yellow. The girth was thirty-eight inches and a half.

It is unnecessary to enter at length into the progress of the case, which was monotonous enough. The main symptoms were cough, nausea, vomiting, and diarrhœa. The amount of urine increased in some degree, but never, as a rule, exceeded about sixteen or eighteen ounces, although on one extraordinary occasion it rose to forty-six ounces in the day.

The belly enlarged slowly, until on the 2nd of October it measured forty-two inches and a half round the level of the umbilicus, when paracentesis was again performed. He was now put upon a course of copaiva. The urine rapidly augmented, often ranging between thirty and forty ounces in twenty-four hours, and sometimes between fifty and sixty. Whether this was owing to the copaiva or not, at any rate the free flow of urine failed altogether to arrest the advance of the disease, although it would appear to have held it in check—so far, at least, as to make life possible and endurable—until November 20th, when paracentesis was again performed for the third time. In the meanwhile, however, it was clear that the man was losing ground from day to day; he suffered much from bronchial catarrh; the urine became uniformly albuminous, its specific gravity ruled low, often it fell to 1010; the pulse at times intermitted, and the main symptom now in the ascendant was debility. The abdomen, indeed, had long been ripe for a renewed tapping, but the patient complained so persistently of extreme weakness, and seemed so instinctively conscious of an utter inability to bear the operation, that for many weeks I declined to sanction it. At last, after a brief show of amelioration in health and strength, he regained confidence, and gave his consent—to his own destruction as it proved. Ninety-six ounces of straw-coloured fluid were drawn off, and for a time he experienced a feeling of decided relief, and passed a good night, but in the morning a great change is reported. He is now stupid and speechless, he cannot be roused to answer questions, neither can he put out his tongue when asked. He is restless, and tosses his limbs about incessantly. On the morning of the 22nd his respirations were hurried, and his pulse almost imperceptible. In the afternoon he died.

Autopsy.—Four pints of amber-coloured fluid were removed from the peritoneal cavity. The peritoneum itself was generally pale and smooth, but somewhat opaque. There were a few bands of adhesion between the liver and the diaphragm, and on the surface of the right lobe of the former a cyst was torn open continuous with the capsule of the organ, and containing a greenish muciform fluid. The body of the liver appeared to be abnormally large. Superficially it was uneven, and everywhere beset with pale-yellow nodular elevations, bordered by intervening bands of opaque and thickened capsular tissue. On section the cut surface presented a multitude of islets of varying sizes, all of a pale-yellow tint, and all separated from one another by fibrous fasciculi. The consistency of the liver was greatly increased. The condensed capsule, as well as the interlobular septa, were freely supplied with blood-vessels, for the most part deeply injected. Spleen much enlarged; kidneys normal in size, unduly firm, and gorged with blood; capsules adherent.

In the thorax there were white patches on the pericardium; the tricuspid valve was unnaturally thick, and measured five inches and a half in circumference. The lungs were highly emphysematous, pigmented, and hyperæmic. The lower lobes were almost airless.

The principal points of interest in the foregoing case are the mode of invasion and the mode of death. People who have to earn their daily bread by the sweat of their brow, or, at any rate, by hard and unremitting mechanical work, are often inattentive to the slighter ailments of life, and this is especially true of drunkards and dram-drinkers, whose normal condition of health is no better than a modification of disease. Now, sclerosis or fibrous overgrowth is one of the most insidious forms of destructive change that any organ ever undergoes, and when it attacks the liver in the shape of true cirrhosis properly so-named, the symptoms of advancing mischief too often pass unnoticed or unheeded. They may, indeed, arouse the suspicions of the sensitive and the delicately-nurtured, but upon the coarser classes of mankind—the people whom I have just described—they seem for the most part to make no impression whatever. Hence, over and over again the history of cirrhosis is only the history of ascites: you cannot by the most searching interrogations trace it back beyond the original appearance of swelling in the abdomen, or in the lower limbs, as the case may be. I say as the case may be for this reason. Of course the distension really begins in the abdomen, but it is not always in that region that it declares itself distinctly to the cognizance of the patient, who may sometimes fancy that it originates in the feet, where it is sure to be recognized the moment it occurs, which is not invariably the case with the enlarging belly, even when it contains fluid enough to compress the cava and make the legs œdematous. This is a matter of some importance in diagnosis, and you would do well to bear it in mind; otherwise you may easily be mistaken and ascribe œdema of the lower extremities to heart-disease or to kidney-disease, when it is really owing to sclerosis of the liver. Enough of digression; I return to the main lines of my lecture. Our own case was most exceptional in the mode of its invasion. I never remember a man in whom the inaugural symptoms of cirrhosis—the precursors of ascites—were so well pronounced and so thoroughly understood by the sufferer himself, as morbid phenomena at least, if not as the forerunners of a dangerous disease.

And now for the mode of dying. Beyond all question I accelerated this man's death by paracentesis. He might have lived a few days longer—possibly for another week—but for that fatal operation; fatal I mean in the date of its performance, which might have been postponed with advantage, but I do not think it could have been deferred indefinitely. I might

and should have waited for a while, but the time was fast coming when paracentesis would be a matter of sheer necessity. Let me dwell upon the dangers of the operation in the case of J. N—. In the first stages of ascites the belly is loose and yielding, and the overcharged vessels of the portal system are free to pour forth the blood-serum in abundance; the tide of osmosis sets strongly outwards from the interior of the veins in the direction of the peritoneal sac, and there is nothing whatever to control its course. At the same time the powers of reabsorption are impaired or altogether annulled. Soon the accumulating fluid begins to tighten the walls of the abdomen and to bring extraneous pressure to bear upon the blood-vessels; the current of exosmosis encounters a check and perhaps reabsorption becomes possible in some slight degree. Still even yet, exosmosis and lack of absorption predominate on the whole over all antagonizing forces, and slowly the ascites gains ground and maintains it. Now, suppose under the circumstances you are induced to tap, what is the consequence? You again set free the veins from the control of the encompassing fluid, and unless they recover their tone rapidly as the amount of urine increases, the tide of transudation flows once more unchecked. The patient is relieved for a season, but for some days he suffers more or less from languor and exhaustion. The drain of albumen is often enormous, and it is impossible the system should not feel severely the large and sudden loss of a principle so essential to its well-being. Now, if the vital powers of your patient are as yet unimpaired, or, at any rate, unbroken, he may endure this loss again and again after repeated operations, but there is always a danger of his succumbing sooner or later. If on the other hand, he is constitutionally weak, or if he is already enfeebled by the disease, then paracentesis becomes doubly dangerous, and the last of a short series of operations may bring life abruptly to a close. Such was the case with J. N—, and that such might be the case I had fair warning. I had no right to put pressure upon him, but manifestly it was a question of time only, and I was misled by his own assertion of strength and competency to stand the trial for the fourth time. We were both mistaken. The man died, not indeed under the operation, but from the operation, not indeed from hæmorrhage, but with all the symptoms of hæmorrhage, as they occur in flooding after child-birth, the ghastly pallor of the surface alone excepted. For the rest the well-known collapse, the sunken features, and the unceasing restlessness and jactitation of the limbs were all

displayed in characteristic form and degree. Clearly the determining cause of death in J. N— was the loss of one of the main life-giving elements in the blood. Nevertheless, it would be unfair to ignore the presence of interstitial nephritis which played the part of a predisponent in the destruction of life. Surgeons, you are aware, have a horror of kidney-disease, and whenever it is proposed to tap the peritoneum, its existence is always an item of evil omen in their calculation of the chances.

It is noted that the conjunctivæ were tinged slightly yellow. Slightly yellowish discoloration of this membrane and a dull earthy complexion of the face present exactly the degree of jaundice which you normally meet with in cirrhosis. Hardly ever do you encounter anything like pronounced profound jaundice. I can only call to mind one unequivocal example.

For the development of icterus in its true colours the occlusion of the bile-ducts must be absolute or nearly so: for the development of ascites no corresponding closure of the portal veins is required; a certain measure of retardation in the blood-flow, far short of interception, will amply suffice. Given, then, the same amount of compression on the portal and on the biliary system within the liver, ascites will follow more readily than jaundice. In cirrhosis, however, the amount of that compression is not the same; the portal veins are mainly implicated in the fibrous overgrowth and sufler more severely than their companion bile-ducts. Moreover, in cirrhosis the proper mechanism of the organ is damaged and the manufacture of bile may well be below the average of health. Strange as it may appear, and in seeming contradiction to the statement I have just made, there is now in Cambridge Ward a man named George P—, who exhibits the very combination which I have affirmed to be so anomalous. He is deeply jaundiced, his liver is undoubtedly cirrhosed, and he has been twice tapped for ascites. Is not this an unequivocal example in point? I am by no means certain that it is so when regarded in its true light. No doubt when we have to deal with an aggregate of morbid phenomena which may be severally referred to a particular organ, it is sound philosophy in diagnosis, it is good policy in practice, to refer all these phenomena collectively to one and the same lesion of that organ—if this be possible. Our first impulse, then, in the case of G. P— would be, if possible, to derive both the jaundice and the ascites from the sclerosis, but, to say the truth, I am disposed to discard the philosophical canon in his case, which is an eminently exceptional one. He was under my care three years ago with

precisely the same signs and symptoms in a minor degree; his jaundice disappeared entirely in ten months; his mother was jaundiced at the time of his birth, and out of a family of twelve brothers and sisters nine have had jaundice in the course of their lives. Probably it has been catarrhal in every instance, but, whatever its nature, with this strange history in view, I am not bound to look upon the granular change in the liver-substance as its all-sufficient cause acting by compression alone. Perhaps it is not going too far to assume that G. P— may have inherited a proneness to biliary catarrh; that this may be the prime and paramount cause, and that the cirrhosis, if it have any connexion with the jaundice at all, may have induced it only or mainly in a secondary way, not so much by constringing the bile-ducts as by arousing the susceptibilities of the biliary mucous membrane—in a word, by calling into play the original sin of the man's constitution.

William L—, æt. 59, was admitted February 12th, 1878. He was a tailor, and a most intemperate man, as tailors too often are. He had been a martyr to winter-cough for ten years, and to shortness of breath and palpitation of the heart from his youth upwards. Moreover, he had suffered from rheumatic fever on three several occasions, and from recurrences of rheumatism in a slighter form many times. A fortnight before admission he found that he could not buckle his belt, and about the same time he observed a swelling of the feet, legs, and genital organs. He coughed much, and his urine was scanty and high-coloured.

On admission.—Face very pale. Considerable œdema of the lower limbs, the conjunctivæ, and even the abdomen, which is enormously swollen, measuring forty-one inches at the umbilicus, and yielding a well-marked wave on percussion. First heart-sound often, but not always, murmur-like at the left apex, and even more so at the ensiform cartilage. Sonoro-sibilant and moist râles throughout the back of the chest. The urine contains one eighth of a column of albumen; specific gravity 1015. Ordered a mixture containing senega, ammonia, and squill, with inhalations of benzoin, and fomentations of digitalis to the abdomen.

February 17th.—Belly tense and glistening; scrotum œdematous; folds between the thighs red and chafed.

18th.—Girth forty-two inches. On the 20th he felt sick, and became drowsy. On the 21st the entire surface of the body was covered with spots, persistent on pressure, and manifestly purpuric. Girth forty-one inches and a half.

Ordered acetate of potassium, with ether and ammonia, in infusion of digitalis every four hours. At the same time five grains of reduced iron were given twice daily.

23rd.—Pulse small, sharp, and thrilling. Tongue dry and brown. Moist râles loudly heard, even by the unapplied ear. Paracentesis performed this day. Over two ounces of blood came away first, then

four pints of straw-coloured serum, the last portion of which was stained with blood.

24th.—In the morning the belly is described as measuring forty-two inches, and in the afternoon as full and fluctuating everywhere. Lies in a state of sopor and semi-consciousness, with slightly stertorous respiration. In the evening the sopor deepened into profound coma, and at 8 p.m. he died.

Autopsy (*abridged from the report of Dr Coupland*).—On opening the abdomen eight pints of dark, blood-stained fluid escaped, and a small sediment of black clot was seen at the bottom of the peritoneal cavity. Spots of ecchymosis were met with in the serous membrane covering the intestines. The liver was adherent to the diaphragm, and in all respects characteristically cirrhotic. The kidneys were granular on the surface, their capsules adherent, and their substance dark-red coloured and tough. They were both below the average in weight, and one presented superficially a large cyst full of a clear straw-coloured fluid. Both lungs were bound to the chest by fibrous adhesions; they were much pigmented, and extremely œdematous and engorged. The lower lobes, on section, were of the deepest red colour—almost black, as if from extravasated blood. The heart was enlarged in nearly equal proportions on both sides. In the right chambers firm fleshy coagula were entangled between the muscular columns. A considerable mass of fat overspread the entire organ, and the muscular tissues were everywhere pale, soft, and flabby. The mitral valve measured four inches in circumference. The aortic valve was thick and nodular, and the aortic lining membrane stippled with atheroma. The brain was found to be unduly vascular.

I have said that J. N— died without hæmorrhage indeed, but with all the symptoms of hæmorrhage save one. W. L— died with hæmorrhage in abundance, but not of hæmorrhage solely, or even chiefly. In the main he died the death of uræmia and chest-disease combined. The symptoms were those of so-called blood-poisoning both before and after paracentesis, although it cannot be denied that the operation helped him on his way and carried him off a few hours before his time. Again, you see, I am responsible for the acceleration of death. When all the arguments for and against paracentesis are duly taken into the reckoning, perhaps I ought never to have operated at all; certainly it was an oversight of mine that in the hurry and excitement of the crisis I forgot the wide-spread purpura and the proclivity to hæmorrhage which might determine an out-pouring of blood anywhere within the organs exposed to it, given the occasion. Now, that paracentesis occasioned the outflow is plainly shown by the sequence of events, which may be divided into three definite and most significant stages. In the first place, we had pure blood issuing bodily from the wound in the walls of the abdomen;

secondly, pure straw-coloured serum from the peritoneal sac; thirdly, from the same source a residue of blood-stained effusion. Beyond the shadow of a doubt, the staining had nothing to do with the blood that flowed on the insertion of the trocar; this idea is at once disproved by the intervening stage of bloodless discharge. It is equally certain, from the same premises, that it *was* owing to the removal of pressure from the surface of the blood-vessels in the peritoneum, as I have explained in the preceding case; serum alone exuding in J. N—, blood and serum intermingled in W. L—. How rapidly this exudation took place, and how soon the punctured sac refilled, I need not remind you. Within twenty-four hours the girth of W. L— amounted to forty-two inches, half an inch more than the last measurement on record before the operation. Let me conclude the parallel between the two cases under discussion. In W. L— the kidneys were far advanced in granular degeneration; his lungs were drenched and gorged with blood, within the vessels in the form of hyperæmia, without the vessels in that of hæmorrhage. It is easy then to understand how W. L— came to die chiefly from blood-pollution. In J. N— it was otherwise; the kidneys in him were only beginning to be granular, and the lungs, though changed, were not disorganized. In him, as aforesaid, the main cause of death was the rapid and ruinous expenditure of blood-elements essential to the life of one whose strength and stamina were far spent already.

A few words remain to be said respecting the propensity to hæmorrhage in cirrhosis. It is not the mere result of mechanical engorgement alone, nor is it limited to the domain of the portal system; its origin is complex and in large measure due to degeneration of the blood and blood-vessels. Hence its not unusual occurrence on the skin and mucous membranes. Remember this whenever you entertain the idea of leeching your patient even in a simple case of jaundice. Never apply the leeches on the loose unprotected surface of the hypochondrium, but over the lowermost ribs, where you may easily control the bleeding by compression. This is one lesson taught us by the study of liver-disease. Another, strongly inculcated by the foregoing cases, is the necessity of keeping a bandage *in situ* long after the operation of tapping, in order to replace the lost fluid-pressure, although possibly Dr Southey's drainage-tube may do away with this necessity. A third lesson—the last and best of all—embodies the pith and substance of my meaning in this lecture. Paracentesis abdominis is far

from being so simple and straightforward a proceeding as it seems, nor is the question to be settled offhand without due deliberation. Of course, on an emergency, when there is imminent peril of suffocation, syncope, or sloughing of the lower limbs, you are bound to tap: there is no help for it; but, emergency apart, I should wait a while and watch the development of the case. I should never tap on the instant, as a matter of routine, for the mere purpose of promoting the action of my remedies. Sooner or later, in the majority of cases, one or other of the perils aforementioned will be forthcoming and then our course is clear. In a minority of cases the disease seems to be at a standstill, neither advancing nor retrograding. These cases are perplexing in practice. I suppose we must operate after a season, provided always there be neither marked debility nor unusual proneness to hæmorrhage to bar the operation. Still I should never sanction the step without grave misgivings. Dr Southey's apparatus may lessen the dangers of debility; I have not often given it a trial, but my experience of it is favourable as far as it goes, and all my colleagues speak well of it. In cases of strong proclivity to hæmorrhage, I should never operate at all if I could avoid it.

LECTURE XVI

A CASE OF INTESTINAL OBSTRUCTION

Delivered July 19th, 1871. Published in 'Medical Times and Gazette,' July 27th, 1871.

A. B—, æt. 51, married, was admitted July 6th, with the following history as recorded in the notes. She has been generally liable to colds; otherwise in good health. Catamenia always profuse until three years ago, when they finally ceased. She then had great pain about the back and umbilicus at the menstrual periods for six months. For the last two years she has experienced at times sudden attacks of pain in the hypogastrium, with distension of the abdomen. These attacks have, for the most part, passed away in the course of a few hours. Considerable emaciation during last six months. For two years the bowels have been habitually constipated, but not specially so during the above-mentioned attacks. About four months ago she was seized with violent shooting pains in the hypogastrium, relieved by the application of six leeches. She remained perfectly well until June 30th, when she suffered from severe pain in the left iliac region, accompanied by flatus, and followed by absolute constipation, which has continued ever since, but there has been no vomiting up to the present date.

On admission.—Face extremely pale and sallow; abdomen enormously distended. Percussion-resonance very imperfect everywhere; dull in left flank when she reclines on that side. Enemata (Oiss) were administered, but returned immediately, before the whole amount was injected. Pulse 120; respirations 20; temperature 99°. 9.30 p.m.: pulse 118; respirations 20; temperature 98·8°. Severe pain at umbilicus; pulse irregular in force. Two enemata ordered, Oj and Oj ʒvj respectively; both returned as before.

July 7th.—Slept after chloral. Pain and griping in hypogastrium and left iliac region. Another enema slowly injected returned immediately before it could be wholly introduced. A slight degree of apparent dulness over flanks when patient lies on either side, and a feeling like that of fluctuation, but not unequivocally the same. Has been vomiting for the first time, eighth day from invasion. Matters vomited simply viscid and bilious. Urine of specific gravity 1025, non-albuminous.

8th.—Pulse 134; respirations 24; temperature 99·7°; no pain, but slept ill; no vomiting; hands clammy; pulse feeble. Seen by Mr Lawson, who discovered nothing of moment within the rectum. 9 p.m.: pulse very weak, 138; respirations 20; temperature 98·4°; severe pain in left iliac region; extremities cold.

9th.—Pulse 136; respirations 20; temperature 98·4°; slept at intervals, but sweated profusely during night; no vomiting; no pain. As much barley-water as the patient can bear to be introduced gently and gradually into the bowel. 4 p.m.: Amussat's operation decided upon after consultation with Mr. Lawson. 5.30 p.m.: enema caused considerable pain, and returned like the rest. Chloroform having been administered, Mr Lawson introduced a long stomach tube eighteen inches into the bowel with the greatest ease. The inference was drawn that there was no obstruction at the sigmoid flexure, and it was decided to defer all operative proceedings. 9 p.m.: pulse 150, very weak; respirations 26; temperature 101·6°; slight delirium.

10th.—Pulse 138; respirations 32; temperature 101°; delirious during night; wandering now and sweating profusely; no action of bowels since June 30th. Seen by Mr De Morgan, Mr Lawson and Mr Hulke. It was decided to give the patient the last chance of life by performing Amussat's operation on the right side. The patient was in an extreme state of collapse when placed upon the operating table. Not more than one drachm of chloroform was administered during the whole operation, which was performed quickly, and without difficulty. At the beginning there was a slight amount of fæcal vomiting for the first time. Towards the close, the pulse became extremely weak, and the respiration slow and gasping. Brandy was injected into the rectum and vagina, with temporary amelioration of the pulse, which soon, however, began to lose force rapidly, while the respiration grew slower and more gasping. Almost immediately after the incision into the ascending colon, and the escape of about three pints of liquid fæces, the breathing ceased.

Post mortem, twenty-four hours after death.—Rigidity marked. *Thorax.* Heart coated with adipose tissue; its muscular walls soft and friable. Lungs emphysematous; otherwise healthy. *Abdomen.* Liver large and fatty. Kidneys normal. There was about an inch and a half of bowel between the caput cæci and the opening into the ascending colon. The peritoneum of both the walls and viscera was smeared over with a creamy yellow lymph, which accumulated towards the surfaces of contact of one coil of intestine with another. The small gut was much distended, and its coils occupied the greater part of the front of the cavity, but below a large blackish slate-coloured coil was seen occupying the hypogastric and left inguinal regions. A thin, semi-transparent omentum passed over the front surface to the lower anterior and left lateral wall; its margin was adherent to the peritoneum, along the left lumbar region external to the colon, and to the front of the abdominal wall, along the line of Poupart's ligament, and the back of the symphysis pubis. The omentum did not reach to the right inguinal region. On cutting off the lower part of the omentum, so as to leave the margin adherent to the abdominal wall, and then removing the greater part of the small intestine, it was found that a fillet of the omentum stretched across from the front wall, near the symphysis pubis, and adhered to the peritoneum, binding down the rectum on the sacrum, as well as to the sigmoid flexure at its junction with the descending colon; and to this spot a coil of small intestine, about six inches from the cæcum, was also adherent. This latter, however, was perfectly free from all obstruction, and only adherent by a very short and broad band, attached to its convex surface. The sigmoid flexure lay quite to the right of the fillet of omentum, and was constricted by it, the upper bend being most tightly gripped, and the

lower—*i.e.* at the point of junction of the sigmoid flexure with the first part of the rectum—much less so, allowing an œsophageal tube to be passed through it, nearly to the upper constricted point. The whole of this coil was prevented from sinking in the pelvis into the recto-uterine pouch, and was in consequence pushed up against the band of omentum, by a large uterus six inches long and ten and a quarter in circumference. The uterus contained many fibrous growths within its walls; it completely filled the whole space between the bladder and rectum, and rose above the level of the brim of the true pelvis. The calibre of the constricted coil varied at different parts from four to five and a half inches in circumference. At the upper seat of the constriction the circumference was one inch and three quarters; at the lower, two inches and one fifth. At both these spots the coats of the bowel were thinned, excepting the sub-mucous one, which was converted into a white shining band. The colour of this portion of the bowel was blackish slate, and between the points of constriction the thickness of the gut was increased, owing chiefly to inflammatory œdema. The descending colon was immensely distended, and gave the appearance at its upper end of being the stomach. Much thin yellow fæcal matter and flatus occupied the bowel. The intestine everywhere was very lacerable.

Gentlemen,—Practically, the first point you have to determine in cases of the foregoing character is the seat of the obstruction. As a rule, you will be fairly well guided in this respect by the routine code of regulations issued for the purpose. Early vomiting, sudden and violent pain, early discharge of stercoraceous matter, scanty urine, an undistended or partially distended abdomen, and rapid collapse are, it is said, the sure signs of obstruction seated in the small intestine, while the converse phenomena, in particular the resonant and voluminous belly, are held to be decisive in favour of the large intestine. Such are the laws laid down by the authorities, and, though not absolutely binding, on an average they will lead you to a correct conclusion. They may be unphilosophical, and it may possibly be that the symptoms are rather determined by the cause and nature than by the seat of the lesion, but you must remember that peculiar lesions specially infest peculiar divisions of the intestinal tube, and are almost appropriate thereto. For most practical purposes, the rules in question may stand, and in accordance therewith we were bound to conclude in our case that the obstruction lay in the lower bowel, where the pathological changes usually discovered on post-mortem examination are strictures and torsions or twistings. Strange to say, at the autopsy we found neither the one nor the other, properly speaking, but an old fibrous band constricting the origin and termination of the sigmoid flexure,

so as exactly to isolate it from the adjoining segments of the intestine. Such bands are common enough in the upper division of the tube, but they are infrequent in the lower. Altogether, the case was anomalous in many respects; the absence of borborygmi and of the well-known serpentine writhing and rolling of the bowel was especially remarkable, and perhaps difficult of explanation, unless we suppose that such movements had taken place before admission, but after that date had become impossible from paralysis or inflammation, or that the obstruction in the bowels had not lasted long enough to produce that hypertrophy of the muscular coats above, which some hold to be the essential condition of visible peristalsis. How are we to treat these cases? I speak of obstructions in the large intestine. Full and frequent doses of opium should be administered, with the view of arresting inordinate peristaltic action, and its consequence, undue distension of the gut, which would only aggravate the mischief. Food should be supplied in small quantities at a time, and in the liquid form alone, and simple enemata of warm water, milk, or gruel should be gently and gradually injected into the rectum, in order to reach, if possible, the seat of the obstruction, and overcome the stricture, or twist, or whatever the mischief may be. If I failed in accomplishing my purpose by these measures, I should, in all cases except those of simple constipation, after the lapse of twenty-four hours, or sooner if the writhing of the intestines should continue unabated, or symptoms of collapse should ensue, deliver the case into the hands of the surgeon, and suggest colotomy—Amussat's operation, as it is commonly called. I should be guided in the selection of the right or left colon by four main considerations—(1) by the *primâ facie* presumption in favour of the left side, which is by many times the commoner seat of the obstruction, (2) by the amount of distension perceptible either in the whole colon or in this or that particular division thereof, (3) by the direction of the before-mentioned writhings and undulations as they seem to bear on a particular point, (4) by the amount of injection which the intestinal tube is capable of retaining, although it must be acknowledged this last criterion is often misleading. Dr Brinton deprecates the use of bougies, long flexible tubes, and the like instruments, whether as dilators of stricture or as vehicles for the introduction of enemata. He even protests against their employment for the purposes of diagnosis. It is impossible, however, to dispense with them altogether, although undoubtedly they *may* do serious damage,

and very often fail in imparting the information we desire. No better example of their fallibility could be adduced than our own case. On July 9th eighteen inches of the stomach tube were passed through the rectum and into the sigmoid flexure without encountering let or hindrance. It was taken for granted, unhesitatingly, but unwarrantably, that the obstruction must be above the sigmoid flexure, and the operation contemplated at the time, with everything ready for its performance—that of colotomy on the *left* side—was at once abandoned. On the following day the operation was performed on the *right* side, just beyond the ileo-cæcal valve. The woman was too far gone, however, for recovery, and she died in the process. I do not mean to say that if the left colon had been opened on the 9th inst., as originally contemplated, she would have lived; but on principle I mention the fact that a day was lost, and in many cases the loss of a day may be the loss of a life. To the best of my belief, no incision of the colon would have saved or appreciably prolonged the life of this woman, even if we had operated on the very day of admission. I cannot imagine that the disorganization of the sigmoid flexure would have been averted or arrested thereby; and even if we had accomplished this great result, there still remained the overlying band of omentum ever ready to renew the mischief. More than this, the walls of the intestine were not simply compressed by the band; they were intrinsically changed, puckered, and contracted. In other words, it was no longer constriction merely, but, to all intents and purposes, virtually, if not nominally, stricture. Let us dismiss now the question of colotomy, which leaves the peritoneum untouched, and turn our attention to the far more formidable operation known as laparotomy, which lays open the peritoneal cavity. This operation has hitherto been limited almost exclusively to obstructions of the small intestine, and I confess I had not the courage to recommend it in my own case. Had I known for certain from the beginning the true nature of the lesion, I should have recommended it; but I did not, and could not suspect the precise lesion discovered, for, as far as I am able to ascertain from a perusal of the cases on record, it was most anomalous and exceptional. The history and symptoms, properly interpreted, seemed to indicate adhesions of long standing and to imply a condition of the lower intestine capable, indeed, for a while of discharging its functions well enough under ordinary circumstances, but capable also of taking on dangerous action and imperilling life at any moment. The slight and fugitive attacks

of pain and distension recorded in the notes would appear to have been rudimentary manifestations of such a capacity; they were only the foreshadowings of that incipient strangulation which was so effectually arrested four months ago, and of that ultimately fatal strangulation which we have failed to arrest. Now, of all cases, those involving old adhesions are the worst adapted for laparotomy. Nevertheless, if the exact lesion had been known beforehand, an exploratory operation, promptly performed on admission, or, at any rate, before admission, might have saved the life of the patient for the time being; for the case was peculiar in the fact that the adhesions were not extensive, but limited to the points of constriction at the extremities of the sigmoid flexure, leaving a large residue of the band entirely unattached as far as Poupart's ligament. If, then, this band had been divided through the free unadherent residue, and the adhesions had been wholly or partially cut away, the constriction might in great measure have been removed, and the bowel restored in some degree to its old integrity of function and its old immunity from disease. Structurally, however, it could hardly have recovered its original calibre. We arrive then at this strange climax of a most extraordinary case. That operation which is deemed most suitable to obstruction of the small intestine might possibly have saved the life of the patient for some time to come; on the other hand, that operation which is our last resource, and often a most valuable resource, in obstructions of the large intestine, here not only failed altogether as it was performed, but would inevitably have failed, however and whenever it might have been performed. As to the origin of the constricting band, I cannot with any assurance of certainty determine its date; it may have been formed four months ago, but I am inclined to make it of much older standing, and to date it from the cessation of the catamenia. Perhaps the peritonitis, in which it originated, may have been connected with the development of the fibrous formations in the uterus. Whether the enlarging uterus had anything to do with the beginning of the mischief or not, certainly it would appear to have accelerated the end by circumscribing the range of the sigmoid flexure from below, and so conspiring with the constrictions above. The false membrane attached to the coil of the ileum may, by a process of constant traction, have interfered with the nutrition and functions of the part; but its influence in this respect must have been insignificant as compared with that exercised by the constricting band.

CASES

WITH

COMMENTARIES

CASE I

CEREBRAL RHEUMATISM

E. R—, æt. 28, was admitted November 13th, 1875, when the following note was taken:—Her father and mother both died young, the mother of consumption, the father of some unknown disease. She herself had an attack of rheumatism six years ago, but it appears to have been slight, and limited almost entirely to the knees. Otherwise her health has been perfectly good, with the exception of a winter-cough. The present illness began about a fortnight ago after a cold. The wrists were first inflamed and then the knees. She has not kept her bed more than four days. There is, however, a vague and confused history of a preceding and far more severe attack, which is said to have occurred one month before admission. In this there was much swelling of the joints, and the temperature ran high throughout. On two evenings it rose to 107°, or to a point between 106° and 107°, but fell again towards morning. She recovered so far at least as to resume work, with some remaining stiffness in one knee.

On admission.—Pulse 132; temperature 100·8°. Complains of pain in most of the joints, but chiefly in the left elbow and shoulder, and in the left hand, which is swollen, red, and tender. There is a feeling of fluctuation in the right knee-joint, and to a smaller extent in the left, but no redness or tenderness. Heart sounds distant, but without definite murmur. No increase in the area of præcordial dulness. Breath-sounds nearly normal everywhere. Urine acid, depositing lithates, but containing no albumen. Tongue slightly coated. Face dingy-coloured, with a purplish mottling in patches. Pulse extraordinarily weak. Ordered a draught containing acetate bicarbonate iodide and citrate of potassium every four hours, along with quinine four grains, and extract of opium a quarter of a grain, twice daily. A senna draught to be given immediately. In the evening, pulse 140; temperature 102·8°—the highest reading. Flushing in face and weakness of pulse continue. States, however, that she feels quite comfortable.

November 14th.—Slept fairly. Bowels freely opened. Morning pulse 120; temperature 99°. In the evening, pulse 120; temperature 100·6°. At this date the pains were almost entirely gone.

15th.—Morning pulse 124; temperature 98°. Ordered brandy, three ounces daily. Evening pulse 120; temperature 97·8°. Sweating copiously. Pupils large.

16th.—Morning pulse 120; temperature 98°. She is now morose and refuses to answer questions, or to show the tongue. The lips are dry and cracked, and the pulse as small and weak as ever. Ordered brandy

and sherry, of each four ounces daily; quinine three grains, with extract of opium, half a grain in pill, every six hours, and a draught of ether and ammonia every six intermediate hours. Temperature at noon 97·6°; at 2 p.m. 98°; at 4 p.m. 97·6°; at 6 p.m. 97·8°.

6 p.m.—Refuses to take her medicine, screaming and struggling all the while. A sixth of a grain of acetate of morphia injected hypodermically.

9 p.m.—Temperature 97·6°. Went to sleep shortly after injection, and is now sleeping and sweating freely. Right hand cold and clammy. At 12 midnight she awoke screaming and struggling as before whenever nourishment was offered to her. Face livid and cadaverous. Temperature 95·1°. Another injection of acetate of morphia, one fourth of a grain. Nutritive and stimulant enemata to be administered when practicable.

17th.—At 2.30 a.m. temperature 98·4°. Enema given and retained. At 4.30 a.m. temperature 99·6°; at 6.30 a.m. 100·6°. Second enema given, but returned immediately. She now appears to be unconscious. At 8 a.m. some brandy was offered by the mouth and in part swallowed, in part rejected with much struggling. When asked to do so, she protrudes the tongue, which is quite clean. Respiration shallow and jerking; hands cold; pulse at wrist imperceptible.

At 9 a.m. she died.

Autopsy, twenty-nine hours after death.—A few thin clots were found in the sinuses of the dura mater. There was very little subarachnoid or intra-ventricular fluid. Centrum ovale of right hemisphere studded with red points, in marked contrast with that of the left hemisphere, which was anæmic. Brain-tissue normal. On both sides the pleural membranes showed vivid injection of the smaller vessels, and here and there some patches of ecchymosis beneath the serous surface. Beyond a few depressions from collapse, limited to the peripheral regions on the posterior aspect, the lungs were perfectly natural throughout. Right side of heart somewhat distended with blood. Pericardium normal. Vessels on the surface of the heart engorged. Beneath visceral pericardium at base of heart were some capillary ecchymoses. The valves were normal in structure. A few pale, fibrinous, adherent clots existed in the right ventricle, but the major portion of the contents were soft black coagula. There was no adherent clot in the pulmonary artery. Left ventricle relaxed. Section of posterior wall, which was in all parts flabby, showed a somewhat confused and mottled appearance in the muscular tissue, but under the microscope nearly all the fibres examined from this region were natural as to striation; in very few were the striæ wanting, or replaced by granules. Liver firm. Lobulation indistinct to naked eye. Under the microscope the nuclei of the hepatic cells were plainly perceptible. Kidneys firm, congested, and somewhat swollen. Spleen in all respects natural.

The foregoing case is full of anomalies from every point of view. Assuming the accuracy of the accounts delivered to us, let us suppose the case constituted of two separate seizures with a brief interval of true convalescence; we then met with the marvel of hyperpyrexia declining spontaneously and

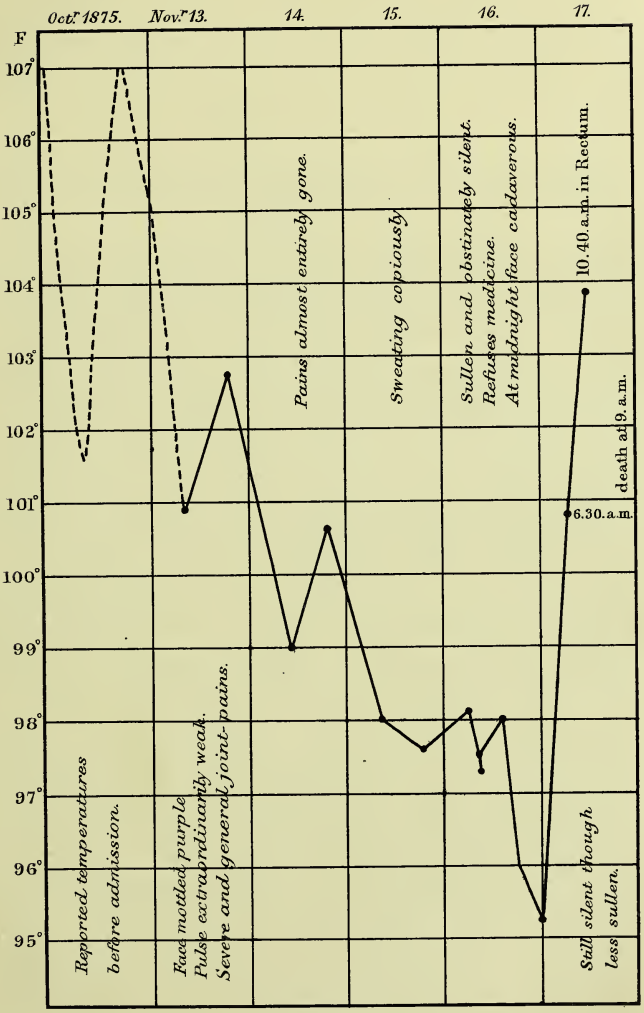
recovery ensuing. This is exceedingly rare in the annals of acute rheumatism, but it is not altogether unexampled. Dr Murchison gives an undoubted instance in illustration. Dr Wilson Fox, indeed, records a spontaneous decline from a temperature of 106°, but the case ended fatally at 103°. On the other hand, let us conceive the entire succession of events to make up one single seizure presenting three several stages, a primary stage and a relapse, with a period of seeming convalescence interposed. We have now no longer to deal with recovery after a spontaneous fall from the height of hyperpyrexia, for on this view the result was death. We have, however, to deal with another and a greater mystery. Are we to believe that the blood-poison, or whatever else originated the brain-shock, lay dormant for a while during the pause in the symptoms in order to reassert its power in a new shape at the close—beginning with hyperpyrexia, and ending, or all but ending, with hypopyrexia? Marvellous as this may appear to be, I am of opinion that it represents the real nature of the case. On any view the great anomaly is the low range of temperature that prevailed on the day before death, when the nervous phenomena were fast coming to a climax. There may, indeed, be what is called collapse in cerebral rheumatism, but collapse with the thermometer at 95·1° is something I should say unprecedented in these cases. True, the temperature rose on the morning of the day of death, but there is no record of a rise beyond 100·6°. Whether or not it ever reached the point of hyperpyrexia it is impossible to say; if so, this must have occurred within the two hours and a half that elapsed between 6.30 a.m. and the time of death at 9 a.m. According to the scheme of registration prescribed, a note of the temperature ought to have been taken during this period, but, as the patient seemed to be dying, the nurse thought proper to disobey orders, and the record is lost. I found, however, that a thermometer passed into the rectum one hour and forty minutes after death marked 103·8°. Now, it has been ascertained that the post-mortem cooling process is slow in the hyperpyrexia of acute rheumatism, while in that of analogous diseases there is, or there may be, a positive rise for some time. On these grounds the chances are perhaps against hyperpyrexia in the present case, but in favour of an increasing degree of pyrexia during the last moments of life. At midnight I ordered a warm bath after the subcutaneous injection of morphia. I ought to have ordered the bath at once as a desperate resource, but the truth is the hand of death was

upon the woman from the day of admission, and nothing in the world would have availed to save her.

One word more. Ecchymoses are said to have been found beneath the serous membranes of the pleura and the pericardium. These are common appearances in cerebral rheumatism; they are common also in all cases of so-called blood-poisoning. Less common, but not infrequent, are fluidity of the blood and softening of the spleen with or without enlargement. Is it possible for cerebral rheumatism to claim kindred with erysipelas, pyæmia, and septicæmia? Is there always a blood-poison to determine the brain-shock which is the essential element in these cases, or may the shock arise directly from the exhausting pain, the worry, and wear and tear of acute rheumatism? Dr Herman Weber raises this question in speaking of hyperpyrexia. May we not raise the same question respecting cerebral rheumatism in all its forms and phases? For my own part I believe in the blood-poison, although I am far from denying that exhaustion and restlessness may help to upset the equilibrium of the nervous system and all the more freely expose the heat-regulating centres to the full force of the poison, whether it cause hyperpyrexia or hypopyrexia.

It might be alleged that the state of the heart had much to do with the anomalies of the case. There were indeed traces of intra-fibrous degeneration, but so slight were the morbid changes seen under the microscope, that it is utterly incredible they could have determined in any degree the mode of dying, or the general character of the symptoms. Even if we were to entertain this view, there would be no grounds for disbelieving in the pre-existence of a poison, which may have determined the degeneration itself.

CHART OF TEMPERATURE. – Emma R.



The interrupted line indicates high temperatures with morning remissions which were said to have been noted a month before her admission.

Whiteman & Bass, *Lith.* London.

CASE II

ACUTE RHEUMATISM, WITH HEAD SYMPTOMS AND A HIGH DEGREE OF PYREXIA

Richard D—, a porter, æt. 19, was admitted into Handel Ward, under Mr Nunn, November 21st, 1878. There is nothing of any moment to record respecting his own history or that of his family.

On admission he gave a most confused and incongruous account of his illness. He had contracted gonorrhœa, together with a sore on the penis; his appetite had been failing for a fortnight, he had not slept for a week, and for the two preceding days he had suffered from pains in the hips, knees, loins, shoulders, and blade-bones—pains at times dull, at times sharp and shooting. Now he complained chiefly of headache, loss of memory, giddiness, and staggering. No trace of any sore could be discovered on the penis. There was, however, some slight scalding, with a good deal of discharge of a yellow colour from the urethra. On the evening of the 24th of November he was seen by the Resident Medical Officer, Mr Fardon, who states that he was distinctly delirious, and that his temperature rose to 103·6°. Chloral and bromide of potassium were given at once, and six minims of Liquor Opii ordered every six hours. On the 25th he was transferred to Cambridge Ward and placed under my care, when the following note was taken. He is a strong muscular lad; the lungs, on physical examination, appear to be healthy; there is no change in the normal area of the præcordial dulness, and no definite murmur there, only a muffling and a slow evolution of the heart-sounds. The bowels are confined, the tongue is dry and densely coated, and the skin hot and harsh to the feel. There is but little discharge from the urethra, and there is neither pain, redness, nor swelling in the joints. He complains of severe frontal headache and pain at the back of the neck, but of nothing more.

It is impossible for me to enter into the particulars of the

sequel. Suffice it to say that the cloud which hung over this man's brain from the beginning never cleared away altogether until December 5th. Throughout the whole of this period on many occasions he was positively delirious, and during his most lucid intervals, when his mind was at its best and brightest, his speech was slow, his understanding dull, and his face blank and inexpressive. Oftentimes, indeed, the face, and more especially the lips, were thrown into vehement contortions, which, when he was awake, added much to his vacancy of expression. Most usually, however, these writhing and twitching movements occurred during sleep. Now and then, when he was asleep in the night time, he was seen to paw the air, a symptom of serious import, and one that ranks high among those which are commonly distinguished by the term "typhoid." I cannot, however, say that I ever felt any starting of the tendons at the wrist, nor were the passages involuntary. Many things were wanting to make up the full tale of the nervous phenomena that characterize the worst cases of cerebral rheumatism. But why were they wanting? Not, to the best of my belief, because there were no elements of danger from the outset, but because we disarmed those elements of all their dangerous power by confronting the malady at every turn, and arresting it, as it were, along the whole line of attack. The same remarks apply to the temperature, which ran high—pertinaciously high—throughout the period aforementioned. We never gave it the chance of rising beyond the limit of 105·3°, the highest reading on record, but that it would have transcended that limit seems a fair inference from the rapid way in which, when lowered by the bath, it rebounded and rose again swiftly, if not steadily, in the direction of a climax.

In all, twenty-six baths were required in the course of thirteen days before we mastered the malady and placed the patient beyond the sphere of danger. In these baths the period of immersion ranged from thirty to seventy minutes. Six times that period amounted to one hour or more. The temperature of the patient on immersion was generally between 104° and 105°; occasionally it was between 103° and 104°. In our own case the symptoms, though characteristic, were not severe, but in many cases it might be inexpedient, or even unsafe, to wait for the higher degrees of elevation here mentioned. The body-heat on removal from the bath ranged from 101·3° to 96·2°. The aggregate fall in the thermometer —the fall during and after the bath—varied from 9·1° to

5·1°; the after-fall from 4·7° to 0·2°. The lowest reading was 95·4°. The temperature of the bath was never above 90° in the beginning and never below 58° at the end. Once the thermometer stood at 74° on immersion. The damp sheets were allowed to remain *in situ* for some time after the bath. This would appear to make a great difference, the temperature often rising rapidly when the patient is put dry to bed, and yet more rapidly when he is well rubbed with flannels. Still, when chilliness and prostration are extreme, we must of necessity have recourse to these measures. Altogether we had a most unmanageable person to deal with. Many times the thermometer, after a steady and uniform rise, all on a sudden shot upwards amain, calling for the bath at a moment's notice. On the other hand, after long-continued immersion without satisfactory progress, the body-heat would suddenly sink to a depth which, to say the least, was undesirable, if not alarming. Once, as before said, it fell to 96·2°, which is far below the mark for the time of removal.

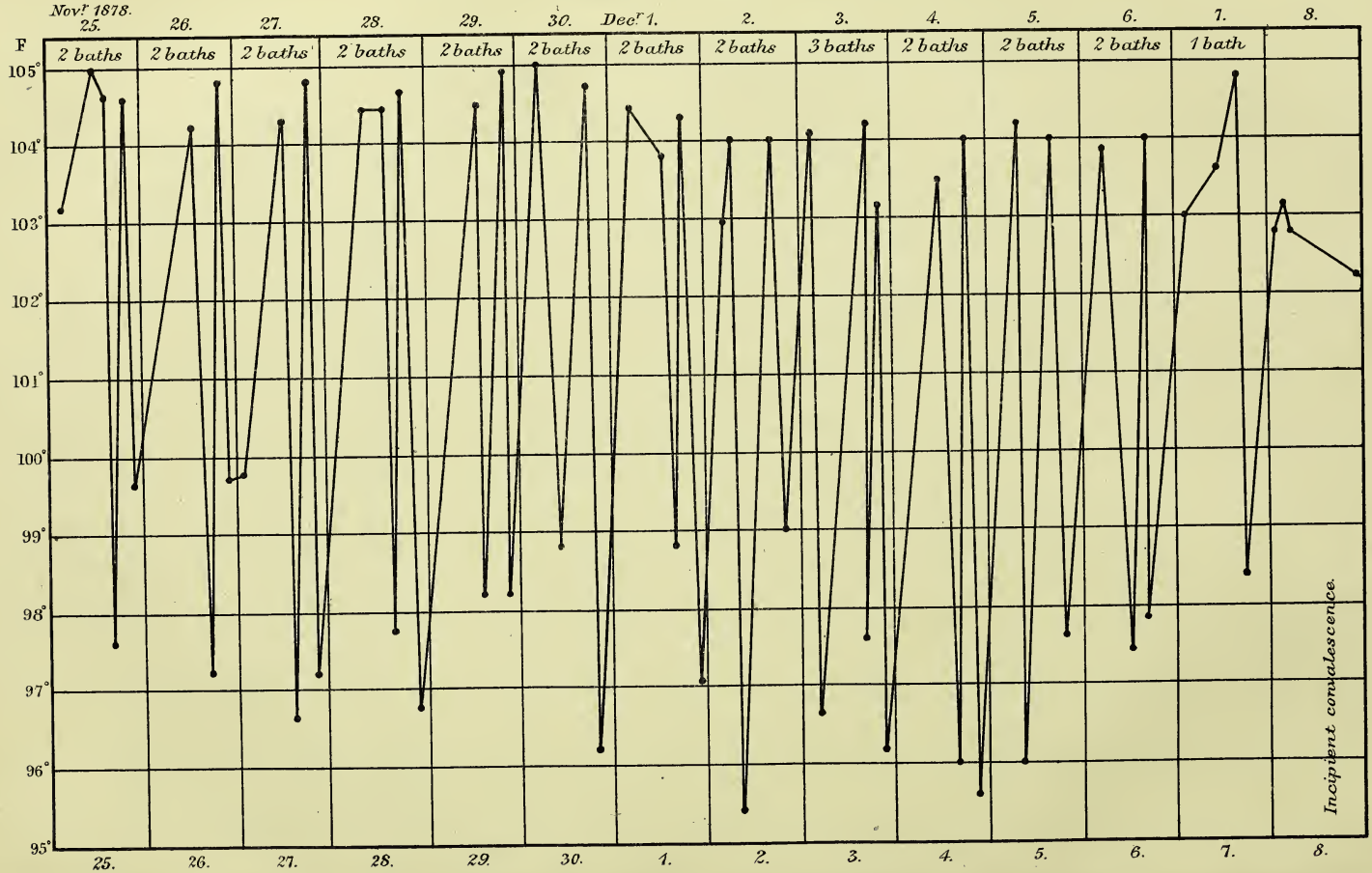
The complications of the case may be dismissed in a few words. There was some slight inflammation of the glans penis from phimosis, and as late as December 28th a trace of albumen was discovered in the urine, probably derived from the last remains of the gonorrhœa. Once there was a threatening of bronchitis, which gave trouble for a time and then passed away, apparently uninfluenced by the bath, as is usually the case. When a man's blood is overheated from within by the operation of systemic forces, he seldom takes cold. On the 30th of November there was an increase in the area of præcordial dulness, and at the same time the auscultatory signs pointed in the direction of pericarditis. On the 4th of December the heart's rhythm is described as cantering. From the date of the last bath, December 7th, the boy gained ground day by day without interruption, unless we regard in that light a bed-sore that formed on the sacrum, or rather, perhaps, I ought to call it a bath-sore, for it is one of the accidents which are apt to supervene upon prolonged and repeated bathing. This is a slight drawback—slight, indeed, in view of the vast utility of the bath—still it ought never to be forgotten; it should be inquired about and provided against, as far as may be, by cushions and other appliances for softening pressure. In our case the sore healed kindly and quickly, but it was followed by a boil on the thigh, showing, it would seem, that pressure alone was not responsible for the damage done, but that water also may have played its part in the process by

coming everywhere into contact with the skin, soaking into its structures, and spoiling their integrity. On the 13th of January the boy was pronounced to be thoroughly well, with the sole exception of a rather rapid pulse and a strong action of the heart. On the 27th of January he was discharged.

I have all along assumed the above case to have been a genuine example of acute rheumatism with cerebral symptoms. Was it a case of acute rheumatism? The boy never suffered from pains in the joints when he was under my care, but he had suffered in this wise before he came under my care, and it is a well-known law, or rather rule, that, when head-symptoms and a rising temperature threaten hyperpyrexia, the proper pains of rheumatism diminish or disappear, whether they return or not. The joint-pains never returned in our patient, but occasionally he complained of pain and tenderness in the back of the head and neck, and once of pain in the arms and the side. Moreover, at one time the physical signs pointed strongly to pericarditis, and towards the close there were suspicions of endocardial change.

It was not a case of gonorrhœal rheumatism: the pains were too wide-spread and too evanescent for that, to say nothing of the intensity of the body-heat and the brain-symptoms. It was not urethral fever: no surgeon would have called it by that name. Was it enteric fever? No physician would willingly have said so; the points of resemblance were far too few and the points of difference far too many. The tongue alone would have seemed to show the falsehood of that view at a glance. Never once, from first to last, did it present the distinctive characters of the typhoid tongue. Again, there were no rose-spots, no diarrhœa, no protrusion of the belly, no chest-symptoms of any consequence, no extreme prostration. One or more of these phenomena might well be absent even in pronounced enteric fever, but that all should remain in abeyance together throughout the course of a fever so intense, is unnatural and incredible. As a last resort, was it pyæmia? Often, very often, pyæmia arises from a primary source of infection within the genito-urinary system, and is known to have arisen from gonorrhœa. True, we never allowed the theomometer a free voice in the settlement of the question; it would have been unwarrantable for us to wait and see how far the fever-heat would rise and fall spontaneously; it was enough, under the circumstances, to know that it rose above the danger-point and to keep it below that level. For the rest there were no positive evidences of pyæmia, which, in all

CHART OF TEMPERATURE. – Richard D.



likelihood, would have put an end to life long before the date of discharge, or even before the date of convalescence.

On a retrospect of all the claims that may be advanced in support of one or other of the above hypotheses, I adhere to my old assumption. I fully believe this to have been a true case of cerebral rheumatism, which would have developed into hyperpyrexia and ended in death but for the measures adopted to arrest its development. In any event, whatever the true nature of the case, the result was no simple recovery; it was an undeniable cure, a rescue from a dangerous disease, accomplished by the bath-treatment, and apparently accomplished by that alone.

CASE III

ENTERIC FEVER, WITH TYPHOID SYMPTOMS PROLONGED INTO A PERIOD OF NORMAL AVERAGE TEMPERATURES

Published in the 'Lancet,' May 9th, 1874.

Edward B—, æt. 39, a cabman, was admitted March 14th, 1874. Three weeks before the date of admission he experienced a feeling of general malaise and languor, and thought he had a cold, but went on with his work as usual. Fourteen days before the same date he was compelled to keep his bed, shivered several times, and suffered from intense headache. During the three or four days preceding admission there had been twitchings of the muscles, and during the last two days delirium, but never at any time diarrhœa; on the contrary, the bowels had been constipated for six days.

At 7 p.m. on the day of admission the pulse was 144, the respirations 44, and the temperature 101·2°; complexion dull and earthy-yellow; marked nervous twitching of the hands, and occasionally of the face; some degree of deafness; pupils small; tongue dry, brown, and fissured; abdomen mottled in appearance and greatly distended; no fever-spots of any kind; no tenderness; area of splenic dulness much enlarged; sonoro-sibilant râles throughout chest; looked livid in the face, spoke feebly, and coughed in a hoarse laryngeal tone. In the evening he passed urine in bed, jerked his knees about, and picked the bedclothes.

March 15th.—Lies in a state of profound sopor. There are sordes on the lips and much subsultus tendinum, with floccitation and carphology. Passed urine and four loose dark-coloured motions under him. Morning: pulse 132; respirations 28; temperature 100·2°. Evening: pulse 128; respirations 28; temperature 99·1°. These observations were taken at 9 a.m. and at 9 p.m. respectively,

16th.—Appears much brighter, and answers questions rationally. There is less twitching of the muscles, and the abdomen is less distinctly mottled than on admission. Morning: pulse 128; respirations 32; temperature 99·1°. Evening: pulse 132; respirations 32; temperature 98·8°.

17th.—Spent a good night. Urine at times passed naturally; specific gravity 1020, no albumen. One yellowish-green pultaceous motion passed in bed. Abdomen tense, but no longer mottled. Morning: pulse 132; respirations 32; temperature 98·6°. Evening: pulse 136; respirations 20; temperature 98·6°.

18th.—Abdomen voluminous, but not tender; motions passed involuntarily, urine naturally. He was delirious during the night, and got out of bed under a strong delusion. Floccitation and carphology

continue. Morning: pulse 140; respirations 40; temperature 99·4°. Evening: pulse 132; temperature 98°.

19th.—Vomited last evening; passed a natural motion of semi-solid consistence; still tremulous, still delirious, and constantly trying to get out of bed; slight carphology. Morning: pulse 132; respirations 32; temperature 98·4°. Evening: pulse 132; respirations 38; temperature 99·2°.

20th.—This morning vomited nearly a porringerful of dark-brown flaky matter looking suspiciously like blood, but no blood-corpuscles were seen under the microscope; tongue dry and brown; great distension of abdomen. Morning: pulse 132; respirations 40; temperature 98·6°. Evening: pulse 144; temperature 99°.

21st.—In great pain, referred to umbilical region; abdomen enormously distended; pulse fuller and steadier; less subsultus. Morning: pulse 120; temperature 98°. Evening: pulse 116; respirations 24; temperature 99°.

22nd.—Speaks clearly for the first time. Morning: pulse 132; respirations 44; temperature 98°. Evening: pulse 120; respirations 28; temperature 97·8°.

23rd.—No subsultus; abdomen has returned to its natural size. Morning: pulse 124; temperature 98°. Evening: pulse 120; temperature 98·2°.

From this time forth convalescence advanced steadily, without let or drawback, except that the pulse ruled high, seldom falling below 100, and never below 90. The patient was discharged April 21st.

The treatment consisted of alcoholic stimuli, oil of turpentine, sulphuric and gallic acids, and quinine in doses never exceeding six grains in the course of the day.

There were on admission many features in the above case in a high degree characteristic of typhus. The dusky, dull complexion, the small pupil, and the mottled surface of the abdomen, which never presented a single rose-coloured spot from first to last, were all of this nature. And yet it was clear in the beginning, from the mode of invasion and from the duration of the disease, and it became clearer every day from the course and character of the ensuing symptoms, that the case was one of enteric fever. The duskiness of the complexion proved in the end to be natural. The main interest of the case centres in the prolongation of the so-called typhoid symptoms far beyond the point which the thermometer alone would have fixed for the commencement, or even for the consummation, of their entire disappearance. On the evening of March 16th the temperature fell to 98·8°; nor was this an isolated example, a chance interpolation in the midst of a higher prevailing average; on the contrary, to all appearance it betokened the coming of convalescence; it stood at the head of a long list of readings, all ranging immediately

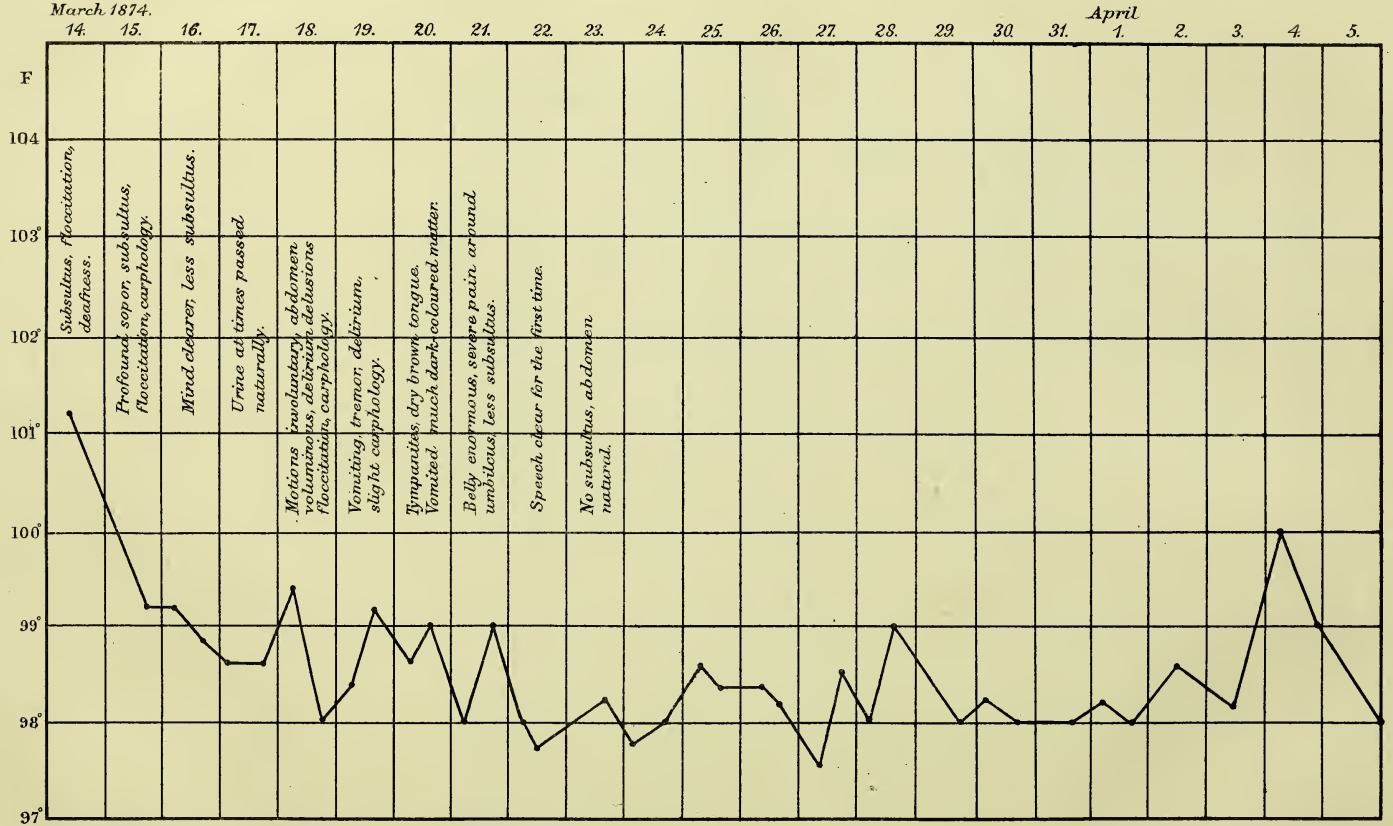
within the neighbourhood of the normal, with deviations above and below that level, but never exceeding 99·4° in one direction or 97·8° in the other. During a series of seven days, reckoned inclusively from the 16th, the average temperature amounted in the morning to 98·6°, in the evening to 98·5°; while during the first four days of the series the average was—for the morning, 98·8°; for the evening, 98·6°. Surely one might have expected a decided abatement in the severity of the symptoms even from the beginning of the period above defined. Some slight amendment must indeed be conceded to the first three days of that period; the stupor ceased, the muscular twitchings declined in frequency, and the urine at length passed naturally; in other respects there was no amelioration at all. The delirium seemed to be worse than ever, the tremors and involuntary passages of fæces continued in full force, while the patient pawed the air and picked the bedclothes just as he had done during the height of the fever, so far as it came within our observation. It was not until the following day, the 19th, that the carphology appeared to be subsiding, it was not until the 20th that the delirium and the tremors came to an end, and it was not until the 22nd (the seventh day in the series) that the subsultus—the last of the typhoid symptoms—disappeared entirely, and true convalescence began. All the while the pulse ran high, maintaining an average of 130. Of course it might be argued that the dark-coloured motions of the 15th contained blood, and that the hæmorrhage kept down the temperature. The fall, however, at the time was insignificant, and the hæmorrhage, if hæmorrhage it was, must have been quite incapable of lowering the body-heat for a permanency. Again, it is easy to say that the observations should have been taken oftener than in the morning and evening alone; and that during some of the long intervening periods the temperature may have reached a far higher level than any I have recorded. This, of course, is just conceivable; but the odds are enormously against it, seeing that from beginning to end the highest ascertained reading never surpassed 101·2°.

On the whole, although it may be heresy to say so in these days, is there not enough in the foregoing anomalies to make us pause before we subscribe to all the dogmas of thermometry? Naturally, the physician looks with confidence on the first fall of the temperature below 99°—at any rate if it occur in the evening—as a conspicuous guide or landmark to direct the course of his proceedings in the after-management of the

case, provided always it be the foremost in a steady series of corresponding registrations. He has only to glance backwards at this date, and he feels himself, as a rule, on fairly safe ground in estimating at any given moment the propriety of relaxing the rigour of the ordinances hitherto enforced. Now, it was altogether impossible for us to feel any assurance of the kind, or to take any step whatever in deference to the date in question, which, in this case, arrived long before it was due; and, if similar cases should accumulate, the value of the guiding date will be seriously discredited. I have no wish to scoff at the cultus of the thermometer simply because it is not infallible or all-sufficient. After all, it was right in the main issue, for the man recovered. It was wrong, however, in its chronology and as a practical guide, and in this respect I deeply deplore its shortcomings.*

* It is possible that the typhoid symptoms may have been prolonged or set up anew by a slight and passing attack of peritonitis.

CHART OF TEMPERATURE. — Edward B.



CASE IV

DIPHTHERIA, PYÆMIA, AND PERITONITIS

CHARLES C—, æt. 17, an apprentice in a large linen draper's establishment, was admitted March 29th, 1879. There is nothing of note either in his family history or in his own antecedents. On the 28th of March he complained of chilliness, followed first by fever and vomiting, and then by soreness of throat, inability to swallow, headache and diarrhœa.

On admission he is described as a strong-looking rosy-faced lad. The fauces are deeply injected, the tonsils are greatly swollen and inflamed, and there are patches of mucus adherent to the surface of each. The glands are enlarged beneath the lower jaws, and on each side of the neck, in both anterior and posterior triangles, but they are not painful or tender to the touch. Temperature 104°; pulse 128.

March 30th.—There is now a small yellowish-white depression, resembling a slough, on the inner side of the left tonsil. The swelling of the tonsils is now extreme; they meet across the median line. Nothing of any moment is discovered on examination of the chest.

31st.—Cervical glands still swollen and now tender. A thick tenacious coating overspreads the back of the pharynx, and on the left tonsil there are layers and dots of muciform secretion. The tongue is loaded with a dirty grayish-yellow fur, and the breath is offensive. The urine contains a trace of albumen, and presents under the microscope pus-corpuscles and epithelial cells, but no casts.

April 1st.—Passed a restless and delirious night, and seems drowsy this morning. No albuminuria now. Nothing can be seen at the back of the pharynx, but there are a few slough-like appearances on the inner surface of each tonsil, and there is a mucous discharge from the right nostril. The breathing is noisy, but there is no laryngeal tone in the voice or cough.

Up to this date the fever ran high; the course of the temperature was steady and continuous, with no great amplitude of range, varying between the limits of 102·4° and 104·4°, while the pulse varied from 112 to 132. On the following day the temperature sank to a minimum of 99·6° and the pulse to a minimum of 96. There was now less tension and swelling of the tonsils and glands, nor was anything apparent on the mucous membranes that bore the least resemblance to exudation or slough.

4th to 8th.—During this period the swelling continued to subside, the tongue cleaned, and the appetite improved. Still, however, although the tonsils looked healthy enough, the back of the pharynx again showed

the same coating as before, and the discharge from the nose became muco-sanguineous, and dried in crusts on the right nostril.

From the 3rd of April to the 8th the pulse ranged from 84 to 96, while the temperature ruled steadily about the normal average, with slight deviations above and below that level.

On the 9th of April the whole character of the disease underwent a change for the worse. The temperature rose to 104·2° and the pulse to 126. The throat became again injected and there was some soreness externally, but no distress in swallowing.

12th.—Glands considerably enlarged. Dark grayish-yellow patches may be seen on each side of the uvula.

13th.—Slept ill; sweated during the night and shivered for a few minutes. The tongue, the limbs, and the whole body are extremely tremulous.

15.—Examination of the throat immediately excites vomiting. Slept well, but had a rigor this morning. Still tremulous, and now complains of pain in the abdomen. Blood and mucus still issue freely from the nose. In the afternoon it was noted that the face had lost its flush and assumed a dingy tint; the pulse was extremely weak and the breathing rapid and laborious, but not laryngeal in tone. The right knee was swollen and fluctuating to the touch.

16th.—Sweated abundantly in the night, but never shivered. Complains of great pain and tenderness over the belly. Vomited this morning, and again in the afternoon. Patch on the left side of the uvula whiter in colour and more sharply defined in shape. Urine had to be drawn off by the catheter. It contained blood, along with one half a column of albumen.

17th.—He is more tremulous than ever, his complexion is more livid and earthy, and the pain and tenderness of the belly are greatly increased. The heart-sounds are extremely weak, but the impulse is distinctly felt. In the afternoon he vomited some grass-green bilious-looking matters, and in the evening was much troubled by hiccough. He is nauseated by everything he takes, and the tongue is dry and black.

19th.—The pain and tenderness, which had abated in some measure the preceding day, were now again intense; the belly was distended; the pulse flagged, the tremors were excessive, and the bowels acted involuntarily. No urine passed naturally; a few drachms loaded with albumen were withdrawn by the catheter.

On the 20th he died.

From April 9th—the day of the crisis, when he underwent the great change for the worse—up to April 15th inclusively the temperature ran a singularly wild and fitful course, the maximum for this period being 105·8°, and the minimum 95·4°. The ranges for the several successive days were as follows :—3·8°., 3·8° 6°, 7·8°, 10·4°, 8·2°, 8·2°. The pulse vacillated also, for the most part in unison with the temperature, varying from 78 to 128.

From the 16th to the 20th, the day of death, pulse and body-heat both alike ceased to rise and fall in this tumultuous way, although they remained irregularly febrile to the last. The breath-rate, however, increased immensely.

Autopsy (*abridged from Dr. Coupland's report*).—The base of the tongue, the faucial arches, and the upper part of the pharynx were all of a bluish-red tint. The pillars of the fauces and the contiguous portion

of the pharynx were coated by a thin layer of exudation, of a brownish-gray colour, not easily detached, and leaving on separation a granular surface. The exudation also coats the whole of the posterior aspect of the epiglottis, and crossing the upper margin passes for a short space over the anterior aspect, showing a sharp line of demarcation there. The epiglottis and the whole of the interior of the larynx are much reddened. Loose threads of false membrane are lodged between the vocal chords. The trachea is throughout deeply blood-stained. Minute ecchymoses lie beneath the visceral and parietal layers of the pericardium. The blood is largely fluid, much escaping from the heart on section of the great vessels. Gelatinous clots are met with on both sides of the heart, but they are not very firmly adherent. The valves present no traces of endocarditis, but on the left side they and the endocardium in general are deeply blood-stained.

Numerous ecchymoses cover the lungs posteriorly. Each lung is the seat of hypostatic engorgement, the lower lobes containing hardly any air and appearing on section of a uniform dark-reddish colour, almost black. The posterior parts of the upper lobes are also engorged; the anterior parts are natural. There are no infarctions and no abscesses.

The peritoneum contained a small amount of muddy fluid mingled with flakes of lymph. Layers of soft greenish lymph occurred over the upper surface of the liver and over the spleen. The coils of the intestine were much injected, The spleen was large, pale, and soft. On the anterior aspect it presented, in two places, irregular pale-yellowish areas, each corresponding to a wedge-shaped mass in the substance of the orpan. One of these masses is encircled by a zone of soft creamy-looking fluid; the other in part pale and fleshy, in part puriform, and reddened by recent blood-clots. The tissue of the organ is everywhere pale and soft. Its weight is ten ounces. Each kidney displays a smooth surface, with some swelling and pallor of its cortical portion. The intestines, full of dark-green semi-fluid contents, offer no notable change in their mucous membranes.

This case is interesting and instructive from many points of view. Singularly chequered in its course and full of vicissitudes and surprises, it yet admits of an easy and natural division into four stages :—(1) The period of invasion with a high and uniform fever-heat and pulse-rate. (2) The period of apyrexia, the delusive break that might have lulled the mind into a false security, but for the fatal change that now ensued in the characters of the discharge from the nostril. (3) The period of pyæmia. (4) The period of developed peritonitis and collapse on the approach of death. The first three were well-defined and sharply set apart from each other; the fourth arose more gradually from its predecessor.

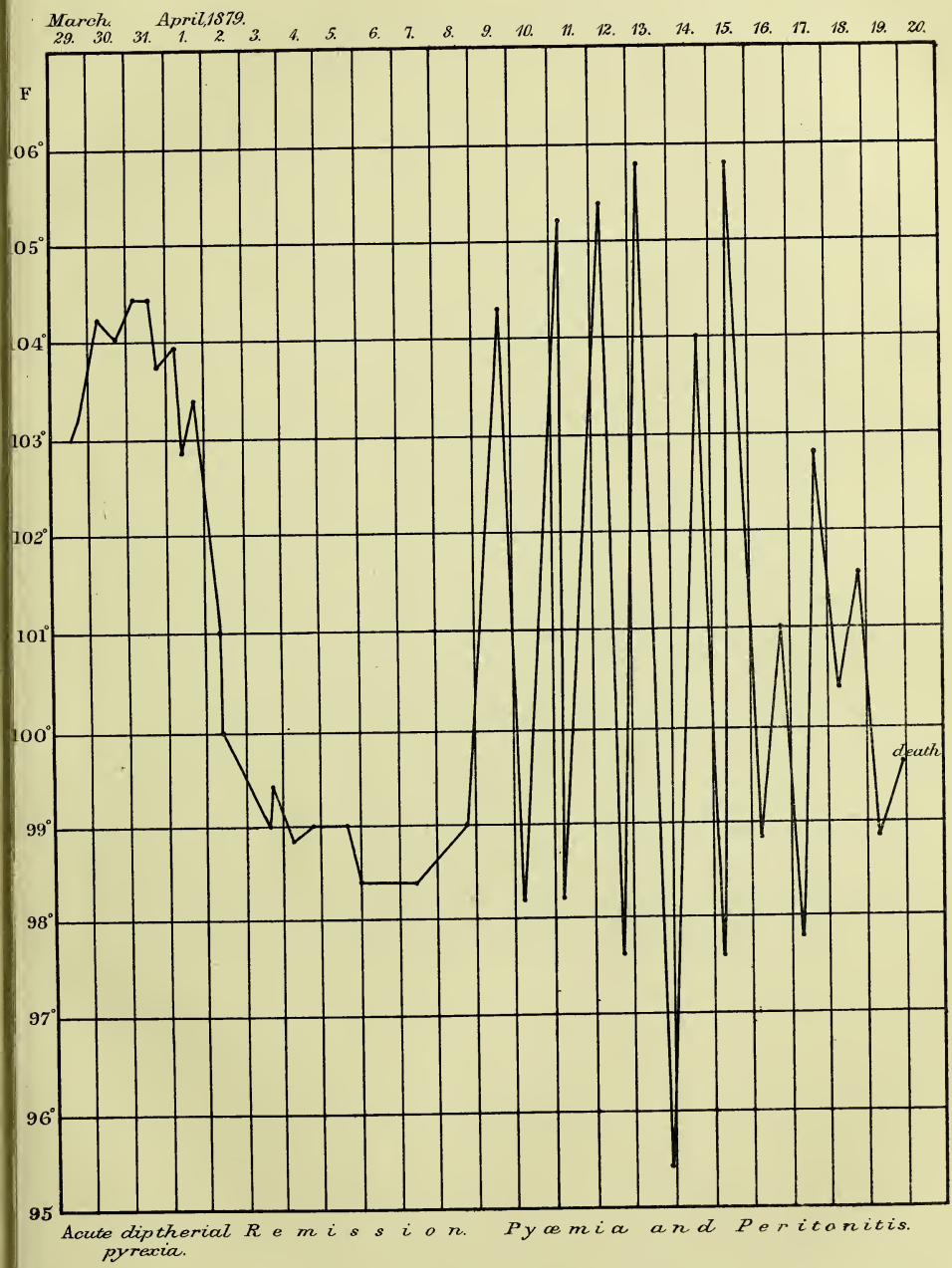
The diagnosis of pronounced diphtheria is one of the easiest problems in clinical medicine. It is well-nigh impossible to mistake the full-grown membrane on the fauces for anything else. But the membrane is not always there, at least in its

maturity. In the case recorded it was undeveloped on the day of admission and remained so for many days afterwards. Nevertheless, diphtheria was all but a certainty from the beginning. Glands may be enlarged in common cynanche, but seldom or never do they reach the magnitude here displayed. The pulse and the temperature may be above the normal average in common cynanche, but seldom or never will they bear comparison with the same phenomena as registered in the present case. Soon the diagnosis became as incontrovertibly certain as anything ever can be in the sphere of medicine. The albuminuria, the renal epithelium, and the thin blood-stained ichorous discharge settled the question beyond all dispute. For all that, even now there was no pathognomonic membrane to be seen on the fauces; the distinctive exudation lay hidden behind the epiglottis, and yet, strange to say, it never fastened fairly upon the vocal chords; there was never a trace of the laryngeal tone in the voice or in the cough.

It is well-known that the far-reaching ranges, the sharp peaks and depressions, on the temperature-chart, even when standing alone, point strongly to the presence of pyæmia. In the case of C. C— the revelations of the chart did not stand alone; they were amply confirmed by paroxysms of sweating and shivering. Indeed, the existence of pyæmia rested upon as good a basis as the existence of diphtheria itself, in despite of its extreme rarity as a sequel to diphtheria.

The course of the fever in diphtheria has been studied by two observers, Faralli and Labadie-Lagrave. They are not altogether in harmony with one another, but thus much they seem to have established. The fever runs high—steadily high—for a few days, reaching 104° or 105° as a maximum; it then falls for a while, and although it may rise again, still in favourable cases it never exceeds at the utmost 102°, but when the case is destined to issue in death, it may mount upwards as far as 103° or 105°. The present case shows some resemblance to the stage of invasion here described, but the ensuing fall is too deep and sudden and the lull in the fever too long. Again, the closing stages were profoundly modified by the symptoms that followed in the train of pyæmia and peritonitis. M. Lagrave ascribes the final ominous ascent on the thermometer to some complication—acute nephritis, lobular pneumonia or endocarditis. He lays great stress upon endocarditis, which exists, according to him, in nearly half the fatal cases of diphtheria. The surfaces of the endocardium, more especially the borders of the mitral valve, are festooned, he

CHART OF TEMRERATURE. Charles C.



Whiteman & Bass *Lithe* London.

says, with minute vegetations, and to this origin he traces the coagula so often seen in the heart's chambers at the autopsy, and not unfrequently evidenced during life by alarming symptoms of dyspnœa and deadly prostration. To the same cause he traces a multitude of local lesions more or less diffused throughout the several organs of the entire system, and more or less stamped with the characters of hæmorrhages, thromboses, embolisms, infarctions, and the like pathological changes. All this is passing strange. To my mind it is beyond the bounds of all conception that the well-known physical signs and post-mortem appearances of endocarditis should have so long baffled the fine ear of the auscultator and the keen eye of the anatomist, ever alive to anything amiss with the heart above all organs, and that not in a few isolated examples alone, but in a whole host of cases. Surely there must be some delusion here, some misinterpretation of morbid phenomena. A proportion of forty or fifty per cent. is simply incredible. It is to be noted that amidst all the variety of alleged secondary lesions resulting from diphtherial endocarditis, M. Lagrave nowhere mentions the main distinctive feature of pyæmia—the presence of abscesses or abscess-like formations. Neither is there any mention of their presence in the latest book on diphtheria, that of Dr Morell Mackenzie; the nearest approach to any allusion of the kind amounting to the statement that in some severe cases of diphtherial septicæmia pus and lymph have been discovered in the arachnoid cavity. Pyæmia proper is indeed rare in diphtheria, but not altogether unexampled; long ago a case occurred under my own care. Dr Herman Weber quotes another, which he himself, however, ascribes more immediately to otitis, the consequence of diphtheria. Drs Wilks and Moxon refer in a desultory way to pyæmia of diphtherial origin. Still, the cases on record are few—so few that the association might easily be forgotten. All the greater necessity then for exemplifying so serious an addition to the ordinary modes of dying in diphtheria.

CASE V

HYSTERIA, WITH CONTRACTION OF THE LOWER LIMBS, ANÆSTHESIA, AND ISCHÆMIA, IN A BOY

Read before the Clinical Society of London, October 26th, 1877. Published in their 'Transactions,' vol. xi.

A. M—, a pale, oval-faced, feminine-looking boy, æt. 14, the son of a French father and an English mother, was admitted into the Middlesex Hospital, May 10th, 1877, with the following history, as recorded in the notes. He has always been a delicate child, and far from strong; but never ailed anything definite up to the invasion of his present illness. Two months ago he began to fall away in flesh. Soon he became low-spirited, lost his appetite, and complained of vertical headache. In the evenings he has been liable to paroxysms of laughing and crying, accompanied by a hoarse barking cough, and by the sensation of a ball in the throat. Occasionally he has suffered from pain in the knees, from palpitation and præcordial distress, and from slight diarrhœa, but never from vomiting. For the last month he has been wholly unable to walk, owing to paresis, rigidity, and distortion of the lower extremities.

On admission.—Urine of specific gravity 1030, acid; contains no albumen and no sugar. The pulse, respiration, and temperature presented nothing peculiar; they were nearly normal now, and throughout the whole career of the case. Voice almost inaudible, but much strengthened by a weak faradic current. Slight dulness, with somewhat bronchial breathing over the left infra-spinous fossa. The legs are firmly flexed upon the thighs, and the feet extended upon the legs in the position of talipes equinus. The genital organs are badly developed for his age. The right testicle is found to be seated high up the inguinal canal, but it can be pushed down into the scrotum; the left testicle is contained within the scrotum, and is about the size of a small horse-bean. Anæthesia and ischæmia were prominent symptoms from first to last, but it is unnecessary to dwell upon them here; they will be fully described in the sequel.

May 18th.—In the afternoon the patient was seen by Dr Sidney Coupland and Dr Thomas Barlow, who have left the following record of their examination :—" The lad was reclining in a chair perfectly impassive and quiet. He answered questions slowly, in a feeble tone of voice, scarcely above a whisper. His appearance was most inanimate, his arms lying by his side, and his features being almost devoid of expression. The pupils were large, equal, and sluggish. No satisfactory

results were obtained from attempts to test the range of his field of vision. His hearing seemed to be naturally acute. The cutaneous and deep anæsthesia before noted was now amply confirmed. He never winced in the least to severe pinches on the cheeks, forearms, and legs from the knees downwards to the ankles. In every case the pinch produced a dead-white areola which slowly faded. The hands were cold to the touch nor did he feel the prick of an ordinary pin, although several deep punctures were made on either aspect of each forearm, sometimes down to the bone, at others far into the mass of the fleshy substance. On no occasion was there the least evidence of sensation, and in answer to direct questions the boy said he felt nothing. It was further noticed that the punctured wounds remained white, and did not bleed. Faradism was then applied to the right forearm, the current being gradually increased in intensity, and although most powerful muscular contractions were obtained, there was no sign of any painful sensation. After the current had been passing for awhile the seats of some of the previous punctures began to bleed, and punctures now made produced bleeding, but the anæsthesia persisted. Whilst the right forearm was being faradized, it was suggested by Dr Barlow that some gold coins should be placed upon the left forearm. Two sovereigns were placed there in the first instance just above the wrist, the lad being instructed to watch them carefully. After they had been there some few minutes without any change in the anæsthesia, another sovereign was added, this being moistened with water, and shortly afterwards a fourth. At the same time the boy was told to place his fingers on the row of sovereigns, and press them against his arm. In the course of about ten minutes the coins were removed, and it was now found that if the forearm were lightly pinched on the extensor surface the boy withdrew it as if in pain. He never responded to pinches on the flexor surface or on the opposite forearm, and the nearer the approach to the exact areas that had been occupied by the coins the more acute appeared to be the painful impression. The pinches were almost immediately followed by a pink flush, contrasting with the dead-white pallor produced by the same means in other parts of the limb. There was the same return of sensation to pricking, and here, again, there could be no question that the sensibility was more acute along the centre of the extensor aspect of the forearm. Moreover, these shallow punctures were followed by free bleeding, while elsewhere the deepest punctures remained bloodless."

19th. 11 a.m.—The boy walked up and down the ward with the assistance of two nurses. He distinctly feels pinches on the back of the left forearm, but nowhere else in the upper limbs.

21st.—In the afternoon he fell into a violent fit, sobbed and gasped for breath, seemed unconscious of everybody and everything around him, and finally, after about ten minutes, burst into tears and recovered. The fits are said to occur on alternate days.

29th.—Great improvement in the sensibility of the face. The conjunctivæ, which were once unimpressible, now react readily to the touch. The extensors of the feet are beginning to relax their rigidity, the talipes equinus is disappearing, and voluntary power over the movements of the toes is returning.

June 2nd.—Strength improving. The forehead and head appear to be insensible to ordinary stimuli. The cheeks, however, and the lower parts of the face show greater sensibility. When the boy's eyes were

shut, and pepper was placed on the tongue, he said there was nothing there, but immediately afterwards detected the taste of common salt.

5th.—This morning Mr E. A. Fardon, the physician's assistant, submitted the lower limbs to examination with the gold test, after duly ascertaining that from the knees down to the ankles they were insensible and bloodless on puncture, however deep the wound might be. The following were the results. It mattered not whether the moistened sovereign was pressed upon the skin by the boy himself or by the experimenter. It mattered not whether the boy was conscious of the presence of the coin or to all appearance unconscious; the result was always the same—sensibility and bleeding in the punctured part. Again, mere pressure with the moistened finger without the interposition of the coin, whether made by the boy himself or by Mr Fardon, gave no reaction. Ice produced the sensation of slight cold; a hot spoon appeared to be absolutely unfelt. Above the knees there is a decided increase in the degree of sensibility. The legs and feet respond, but not violently, to a strong faradic current.

7th.—Mr Fardon pursued his examination of the lower limbs after determining the pre-existence of analgesia and ischæmia as before. No difference was ever discovered between the two legs in any case. Sometimes the boy's attention was concentrated upon the experiment, at others it was drawn away from the proceedings, which were often conducted out of sight and underneath the bed-clothes. In some cases, the several substances were carefully wrapped in paper, the wrappers being at times visible to the boy, at times invisible. In no instance could any difference be made out in the result, neither did it appear to be of any moment whether the boy himself held the objects on the skin, or whether they were pressed upon by another; nor, again, whether they were moist or dry on application. The experiments with the gold coins on the whole corroborated the evidence of former trials. Similar experiments were essayed with silver, bronze, and wood severally, but without success: no sensation accompanied the puncture; not a drop of blood flowed from the wound. Sometimes, but most exceptionally, the gold coin failed to act, or acted imperfectly; the remaining substances never acted at all.

8th.—Half-an-hour after midnight, when the boy was asleep, a pin was pushed through the bed-clothes beneath the skin over the tibia. The boy started, withdrew his leg with an expression of pain, and seemed disposed to cry. The blood issued freely from the wound.

For some days from this date he suffered first from bronchitis, with marked exaggeration of his brassy cough, and then from a rather diffuse but not intense attack of rheumatism. After a brief interval of returning health he was seized with diarrhœa, which, though severe, made no great impression upon him. He soon gained strength, his voice improved in power, and he walked about with comparative ease.

July 4th.—This morning, as he was engaged in the composition of a poem wherein his whole mind seemed to be absorbed, the opportunity was taken to plunge a pin into his leg. He never appeared to feel it in the least, nor was there any bleeding from the wound.

10th.—Again he suffered from bronchitis—this time more severely than before, and for a longer period.

27th.—Mr Fardon renewed his experiments, with the assistance of Mr Laybourne. It was first ascertained that the lower limbs, from the knees down to the ankles, were insensible and anæmic to deep punctures.

The thighs, however, and the feet retained their normal sensibility and capacity of bleeding. Again, as before, concealment of the coin made no difference in the result. Again, exceptionally, the gold failed, but almost invariably succeeded in provoking pain and hæmorrhage. On one occasion a gold ring was used; it was then found that the reaction was limited to the zone of pressure, and that no change occurred within the circle. Once the boy pressed a sovereign on the skin in the belief that it was a shilling. No reaction ensued. Afterwards, when the same coin was given to him, and he was shown that it was a sovereign, he placed it upon a distant part; the usual result followed its application.

Hitherto silver and copper coins had proved to be inoperative. The experiments had been mainly conducted upon the right leg, and now it was noticed that sensibility and bleeding were apparent where a shilling had been placed; but, on further investigation, it was discovered that the whole leg, as before defined, had become more or less sensitive, especially but not exclusively over and around the parts experimented upon. Attention was now transferred to the left leg, which remained obstinately insensible, and coins were applied—gold, silver, and copper—carefully wrapped in paper; so carefully indeed that the experimenters themselves were unable to name the coins enclosed in the several wrappers. Moreover, the envelopes were often concealed from the boy's view. If then he could form any idea of their respective contents he must have been more than clairvoyant. The sensibility and the bleeding that followed the puncture were entirely confined to the part pressed upon by the gold coin.

August 4th.—After the usual preliminary test, the lower limbs were submitted to a final examination with the coins; all the subjoined experiments, with the unavoidable exception of the two first, were conducted both openly in view of the patient and secretly under the bedclothes, without any difference in the result. In every case the coins were allowed to remain *in situ* for ten minutes. All the experiments were repeated, and in every instance but one the issue was the same.

A shilling was pressed upon the skin, the boy being told it was a sovereign. No reaction took place. A sovereign was laid on in the same manner, the boy being told it was a shilling. Sensation and bleeding followed. Gold, silver, and copper coins were applied in various ways, in sight and out of sight, moist and dry, covered and uncovered, wrapped in paper beneath an envelope of silk, and wrapped in silk alone. The spaces corresponding to the gold bled everywhere, with all the signs of sensibility present, and without a single miscarriage. The silver spaces remained insensible and bloodless with one exception. At the close of a protracted manipulation, embracing a large proportion of the limb, the part compressed by a shilling proved to be sensitive, and bled. The copper coins were inert from beginning to end.

14th.—There is dulness on percussion with bronchial breathing over both upper lobes posteriorly, and in the left front impaired resonance, with deficient expansion and blowing respiration.

14th to 24th.—Throughout the whole of this period sensibility and capacity of bleeding were shown by numerous experiments to be normal everywhere.

25th.—Left the hospital for the Convalescent Asylum at Eastbourne.*

* Came back to show himself in the spring of 1878, when he was perfectly well.

The foregoing is a case of hysteria, pure and simple in the best sense of the term. The boy is thoroughly truthful, straightforward, and uncomplaining. How then came he to be hysterical? Many things point in this direction. He is a precocious boy, he is a poet of no mean order for his age; he is singularly well-bred, gentle in manner and speech, far beyond the mark of his condition in life. All these items deserve to be taken into the reckoning: they evince a fineness of organization, bordering closely on the hysterical frame of mind. Again, he has lived under the dominion of an over-fond mother, who has done her best, or her worst, to spoil him. Finally, his sexual organs are ill-developed. Probably this is the main element in the predisposition. As for the phenomena of the case, the following are the most salient features:

(1) We have paresis with rigidity of the limbs—contracture, as the French call it. Paresis in hysteria is common enough, and contracture as a passing symptom by no means uncommon. In the permanent form, it is exceedingly rare. Charcot and Briquet, in all their enormous experience, record only fourteen cases. In our case the contracture has disappeared, but there is still a trace of the paresis remaining.

(2) We have anæsthesia amounting to analgesia. As a rule in hysteria, loss of sensibility, when widely diffused, takes the form of hemianæsthesia, but in this respect our boy is a law unto himself; in him the distribution of the insensibility was unique and even grotesque, setting at defiance all precedent and all anatomy. Stranger by far is the third feature, the ischæmia, or stoppage of the blood-flow. Strangest of all is the last, the influence of the gold coins. What is the meaning of this influence? Abroad, at any rate in France, electrical, electro-chemical, or electro-physical theories appear to be in the ascendant. On the other hand, morally regarded, metals, at least polished metals, are attractive things, and in virtue of this property, when laid on the living surface, they may engage the imagination and hold fast the attention, thus recalling warmth, circulation, and sensibility to the cold, bloodless, and insensible regions of the body. Unfortunately for this view different metals are necessary in order to act upon different individuals. Now, we have a fair idea of the laws of attraction in metals, so far as they severally sway the bent and bias of unhysterical mankind. Gold, for example, in ordinary life, is far more fascinating than copper. Why then should the charm of gold ever yield to the charm of copper, as apparently it *may* do in hysteria? Is this idiosyncracy, or

is it one of the many caprices and contradictions of the great neurosis? Again we are all abroad, and the solution of the mystery seems to be as far off as ever. The moral theory finds favour with Dr Russell Reynolds. One of our experiments may be adduced in support of this view. Once when the boy believed the sovereign to be a shilling, no reaction ensued. It must be borne in mind, however, that the sovereign, when known to be gold, sometimes failed, though very seldom. Moreover, the experiment, when tried a second time, with every precaution to exclude error, gave the opposite—the usual result. Other experiments, many times repeated with the same unvarying issue, tell uniformly against the moral hypothesis. If there is no lurking fallacy here, if the boy really had no consciousness, and no suspicion of the presence, the pressure, and the nature of the coins, when carefully concealed from his view—above all, if in his last and best examination with the coins he fully believed the shilling to be a sovereign and the sovereign to be a shilling, when he was falsely told so—the moral theory cannot be maintained to the exclusion of all others. Imagination may go a long way in many cases, but clearly imagination is not all in all in every case and on all occasions. Probably it had nothing whatever to do with our own case *as far as the coins are concerned.* Again, Dr Reynolds ascribes the insensibility itself, and apparently also the ischæmia, to mental and moral agencies. He even ventures to ask whether the anæsthesia may not exist only at the time of observation. The experiment that we performed when the boy was asleep speaks strongly in confirmation of this idea. He felt the puncture most keenly, and the blood flowed freely when he was taken by surprise, and when there were no impressive surroundings, no old associations to call forth the phenomena. Possibly here Dr Reynolds has hit the nail upon the head. The experiment, however, was only once tried: it was thought unwarrantable to repeat it.

As for the few failures of the gold, and the still fewer successes of the silver, in all likelihood they were mere accidents in the main. When the gold failed, the time of application was guessed only: it never failed when the time was duly prolonged and accurately measured at ten minutes. Again, the silver never succeeded, save when at the close of a long series of experiments the entire limb from knee to ankle had become more or less charged, if not everywhere saturated, with the gold. Nevertheless, if we may judge by comparison

with the copper coins, which never acted at all under any circumstances, it is possible, but by no means certain, that the silver may have exercised some slight auxiliary power, just so much as to reinforce the gold and to consummate the result over areas insufficiently excited by that metal, but not so much as to determine the result by its own unaided operation.

Whatever the *modus agendi* may be, the average duration of the forces in play is perhaps undetermined as yet, but it seems to be long enough to give fair promise of utility in practice. Coins and metallic bands or plates have been employed remedially in France on a large scale. In our case they have been put to no practical use. Faradism alone has been applied along with the administration of shower-baths, valerian, cod-liver oil, and iron.

CASE VI

DIABETES TREATED WITH OPIUM

Read before the Clinical Society of London, April 28th, 1871. Published in their 'Transactions,' vol. iv.

At a time when the value of opium in the treatment of diabetes is a subject deeply interesting to the profession at large, and much discussed among our own members, perhaps I may be at liberty to lay before the Society the following case, bearing as it does, though somewhat equivocally and indecisively, upon the point in question. I have only sketched it; the whole case is far too long, and for the most part too monotonous, for detailed description. It is interesting on this ground: it was submitted to a fairly severe course of opium, and it improved; whether it improved in consequence of the opium, that is the problem I am desirous of propounding to the Society for solution.

Samuel P—, æt. 35, formerly a stout strong man, first engaged as a professional bowler at cricket, then as a fitter of lightning-conductors, and occasionally as a bricklayer, had been in good health until thirteen years ago, when he contracted gonorrhœa. Nine years ago he was laid up for ten weeks with "influenza," which was attended with great prostration of strength. For the last two years he has been losing flesh; nineteen months before admission into the Middlesex Hospital he had an attack of "lumbago," and since that date his appetite has been increasing. One month after the lumbago, eighteen months before admission, he first noticed that he began to make more water and more frequently than usual. During the last six months before admission his lips and teeth stuck together in the morning, and a viscid substance exuded from the roof of the mouth. At the same time his tongue began to darken in colour, and in three months it became quite black. For seven or eight weeks before admission he perceived a peculiar taste in his mouth, sometimes like sugar, sometimes like rotten apples, on other occasions like various other things. During the same period his sight grew dim, and he was often obliged to give over reading in consequence of the lines becoming indistinct. Shortly after admission, Mr Hulke examined his eyes with the ophthalmoscope, and reported "no opacity of the ocular media and no appreciable defect of the retina, but manifest

hypermetropia." The dimness of vision then would appear to have only a secondary connection with the diabetes.

On admission, November 6th, 1869, he presented himself with a foul, densely-coated blackish tongue, a feeble stooping gait, and an expression of abject despondency—altogether as graphic a delineation of the diabetic aspect as one would wish to see.

Before treatment the urine in twenty-four hours measured 8 pints 7 oz. Specific gravity 1042·5. Total amount of sugar in twenty-four hours 6095 grains.

During treatment the proportion of urine and sugar and the specific gravity varied within considerable limits. The highest recorded amount of urine was 11 pints 18 oz.; the lowest 4 pints 10 oz.; the last amount occurring on January 29th, 1870, long after the full course of opium was over. The highest reading of the amount of sugar in twenty-four hours was 6600 grains on the last day of December, the day before the omission of the full opium treatment; the lowest 1914 grains, on January 30th. These amounts were calculated by multiplying the loss of density after fermentation by the number of fluid ounces passed in the day; one grain of sugar in a fluid ounce of urine exactly corresponding to one unit of lost density, according to the curious and most convenient law discovered by Dr Roberts of Manchester.

The highest specific gravity was 1047 on December 24th, 1869, and the lowest 1032 on December 3rd.

The principal symptoms over and above the condition of the urine were a severe and persistent headache, pains in the loins, intense thirst, ravenous appetite, hæmoptysis once at least, if not twice, cough, night-sweats very profuse, sponginess of the gums and looseness of the teeth, languor, debility, and on one remarkable occasion overpowering drowsiness lasting for many days, always aggravated by the opiates administered, and culminating with the acme of the opium treatment, though continuing in a modified form for some time afterwards.

When I speak of the opium treatment I mean the period when he was taking the drug in large and repeated doses, extending from November 15th to December 31st. Before and after this period more or less of opium was prescribed all along in ordinary doses. Other medicines administered at various times were bromide of potassium, creasote, belladonna, quinine, sulphate of iron, nitro-hydrochloric acid, cod-liver oil, bicarbonate of potash, and the citrate of iron and ammonia. The amount of opium given and the mode of its administration will be shown in the remarks appended. The diet and regimen throughout were those usually assigned to diabetes. The progress of the case fluctuated for a long time. No unequivocal improvement occurred until January 14th, a fortnight after the omission of the opium treatment, and a month before the date of discharge. The quantity of urine and the specific gravity were recorded daily; the amount of sugar excreted in twenty-four hours was taken repeatedly at convenient intervals. I have not, however, thought it desirable to encumber the case with the whole of these details. From January 30th, the date of the lowest amount of sugar, until February 12th, the day of departure, there are no more records of the sugar excreted; the urine at the last-mentioned date measured 4 pints 12 oz., and its specific gravity marked 1037. During this period the man continued to gain in weight, and in the end left the hospital with a fair portion of enjoyable health and strength.

He was discharged summarily for disobedience to orders — a very common event in the history of diabetes.

When first I saw this case I at once gave it as my opinion that I should do it no good whatever. Diabetes and death were to all appearance written in the man's face. The obvious emaciation, the slowness of movement, and the aspect of acute melancholy, all betokened at a glance a difficult and even desperate case. To make matters worse, if possible, we found in the progress of the malady that the patient coughed, and once or perhaps twice spat blood. Moreover, he sweated profusely in the nights, and sweating is a most exceptional symptom in diabetes. Altogether the case seemed on the point of terminating in one of the usual modes of dying in diabetes—by a variety of pulmonary phthisis. This suspicion was only corroborated by physical examination, which revealed evidences of condensation in the upper lobes of the lungs. Furthermore, there were symptoms which are held in an especial manner to bar the administration of opium; in particular, drowsiness and severe persistent headache. Nevertheless I resolved to make the experiment within certain limits, and to give the opium a fair chance. It was exhibited first in the form of the compound soap pill, then in the form of morphia every night and in gradually augmented doses of Battley's solution by day; the ultimate amount of the solution reaching 144 minims daily, or about the equivalent of 12 or 14 grains of opium, the morphia making about one grain more. I might have gone beyond this, but the headache increased in intensity, and the drowsiness became so overpowering, and at the same time so unequivocally aggravated after each dose of the opiate, that I had not the courage to proceed, but at once reduced the amount of opium to 5 grains of the compound soap pill each night. It must be acknowledged that I had disregarded these symptoms, when less severe, at the beginning of the treatment, and that they disappeared or abated at times even during its continuance. It must be acknowledged that the pupil was large, or at least above the diameter of average pupils, and some people I believe would have set the symptoms at defiance and run all risks. This, in my judgment, would have been indefensible: so I abandoned the opium—all but a fraction of what had been taken before.

Now mark the sequel. The urine at once diminished in quantity, the specific gravity remaining about the same. This diminution in quantity continued with more or less

oscillation up to the day of discharge. The amount of sugar also began at once to decline, and in the end it was immensely reduced. On the other hand, the general symptoms underwent no improvement until January 14th, a fortnight after the full course of opium was abandoned; in particular, within the interval beginning December 28th and ending January 15th, the weight had fallen 7 lbs. After January 14th the man improved wonderfully in general health; he lost altogether the distinctive diabetic expression, and his appearance, though pale and delicate, was not that which would forcibly convey the idea of any incurable disease.

What has opium done in the case before us?

We are aware that certain medicines, when continuously administered, after a time arrive at a climax and give rise to symptoms of incipient poisoning; they are then, of course, discontinued, or the amount given is very minute. Mercury, for example, is administered in syphilis until the mercurial action culminates in an affection of the gums. It is then discontinued or materially reduced; the syphilitic symptoms generally or often disappear, and their disappearance is assigned to the past operation of the medicine, or, more correctly speaking, to the strong and abiding impression originally made by the medicine on the system, an impression continued after the discontinuance or diminution of the remedy. The same thing may be said of arsenic after the production of the dry throat, the silvery tongue, and the œdematous eyelids. The same thing may perhaps be said of quinine, when it produces deafness and ringing in the ears; the same thing perhaps of iodine, when it gives rise to catarrh from the eyes and nostrils; and perhaps the same thing of belladonna, when it culminates in causing dryness of the throat and disorder of vision. After the climax the medicine is abandoned, the impression abides: that is the conclusion or the assumption.

On the other hand we often reason in a wholly different way, on the well-known principle, "Sublata causa tollitur effectus." When, for example, after the occurrence of objectionable consequences, we throw overboard a medicine, we are in the habit of assuming that we throw overboard along with it all its medicinal operation, and that any ensuing amelioration in the symptoms is due, not to the still-abiding influences of the remedy, but to the entire abolition of those influences, to the aid of time, or to the operation of other remedies. How are we to reconcile these two processes of

reasoning? This is a serious question, and strikes at the root at the whole system of medical logic, if we are at liberty to draw opposite conclusions from premises apparently the same.

One would naturally say that where recovery or marked amelioration after a climax rapidly followed the discontinuance of a remedy, it was all owing, or might be owing, to the original impressions of the remedy now discontinued; but where there was either no recovery at all, or only a long-delayed recovery or amendment, we should then hold that the original treatment had either done no good, or done positive harm.

To return to our own case; are we to imagine that opium has a climax like that of mercury, and that when discontinued in consequence of reaching that climax it still continues to do good? I can hardly imagine anything of the kind in the case of our patient. In the first place, I am not sure the phenomena of the presumed climax were altogether due to the opium. In the second place, if they were so due, opium is one of those remedies which we should expect to do good, and to declare their influence for good, before they have reached the point of incipient poisoning. Now, it is true that shortly after its discontinuance there was considerable diminution in the volume of the urine, and a considerable fall in the amount of sugar; but then on the two days last preceding that discontinuance the sugar had enormously increased, and on the day immediately before, the amount stood at 6600 grains in 24 hours, the highest reading recorded. Moreover, there was no unequivocal sign of amendment in the general health until after the lapse of a fortnight—an interval characterized by depression, debility, and emaciation. The interval, in my opinion, is far too long for the hypothesis that the climax of the opium treatment constituted the turning-point of the disease. I should rather conclude that the opium treatment, if it did no harm, at all events did no good, and I should ascribe the improvement ultimately ensuing to other remedies, to a regulated diet, and to favourable sanitary conditions in general.

Perhaps the alkalies prescribed towards the close may have done good service; for my own part I have a stronger belief in the efficacy of quinine, iron, and cod-liver oil, all of which I administered freely.

CASE VII

CARIES OF THE TEMPORAL BONE, CEREBELLAR ABSCESS, AND FACIAL PARALYSIS

Published in the 'Lancet,' February 12th, 1876.

P. S—, a baker, æt. 24, was admitted on the 6th of July, 1875, when the following note was taken. There is nothing of any moment in his family history, and nothing whatever that bears upon his present illness, except that a brother is said to have died at seven years of age from "inflammation of the brain." He himself, twelve years ago, had scarlet fever, and about four years ago gonorrhœa. His hair has been falling off for the last twelve months, but he denies ever having had a sore-throat or a rash upon the skin. He has had otorrhœa from childhood, and has often complained of severe pain extending from the ear downwards to the shoulder and upwards to the scalp, the pain being most intense over the temples on both sides. His present attack commenced three weeks ago, with headache, vomiting, giddiness, and shivering. He found himself unable to whistle; the room appeared to roll round; and he could not voluntarily close his right eyelids.

State on admission.—Face expressive of languor and distress; utterance thick; mouth distorted to the left on speaking. The powers of smell and taste are good, and vision is unimpaired. He is deaf, however, in the right ear, and cannot hear the ticking of a loud watch when placed upon the pinna. The tears escape from the right eye in abundance. There is pain in the temples and forehead; pain also, chiefly nocturnal, over the right mastoid process. An offensive discharge of pus and blood flows freely, with short intermissions, from the right ear. The right orbicularis palpebrarum acts imperfectly; at times, however, it just closes the eyelids, at other times it fails altogether, and leaves an interval of one or two lines between their margins. Its action is always strongest when the corresponding muscles of both eyes are called into play in unison. No cough; no heart-murmur; no tenderness over the mastoid process; no apparent paralysis of the limbs. Pulse 68, respirations 20, temperature 98°.

July 7th.—The right eyeball moves outwards in a feeble and jerking manner, and the cornea scarcely reaches the external canthus at all.

10th.—Pain relieved. On attempting to raise the eyebrows the surface above on the left side is furrowed in the natural way, the right side is simply blank and smooth. A similar difference is seen in the vertical furrowing when he is desired to knit the brows. On showing the clenched teeth the left aperture of the mouth is widely and irregularly opened; the right remains closed, or nearly so. When he is told

to shut the eyes by a forcible effort, the right lids just meet and no more, while the left are thrown into wrinkles and compressed strongly upon the eyeball. When the left eye is held open and he is requested to shut the right, he is wholly unable to do so, the unclosed interval measuring two or three lines. Even when the left eye is held shut by the finger and he is desired to close the right, the interval is quite distinct.

12th.—Pain across the forehead again severe; there is pain also over the entire dome of the cranium above the level of the ears.

14th.—Still in pain and extremely restless. The features are now more evenly balanced, and the naso-labial sulcus is less deeply cut on the left side. He closes the right eye when the left is held shut, even to the extent of corrugating the lids, and shows the teeth fairly well, with only slight distortion in the act. The right external rectus, however, is still wavering in its movements, and fails to bring the cornea home into the outer canthus.

15th.—Appears more drowsy than heretofore, and complains more of his head. Wandered about noon. Pulse 52, feeble and flagging. When he raises the eyebrows the left forehead is conspicuously wrinkled, the right in a lesser degree, but quite distinctly. He is, however, again unable to close the right eye when the left is held shut. In the evening a minute injection of morphia was administered, and he slept fairly during the night, though waking and wandering at times.

16th.—Morning: pulse 72, respirations 16, temperature 98·6°. Right pupil rather larger than left. There is now considerable distortion of the lips in the act of showing the teeth. The centre of the cornea remains fixed in the median line, and cannot apparently be moved outwards. This observation, however, must pass for what it is worth. It is by no means certain whether the patient thoroughly understands the instructions given to him. He is at best but half conscious, although suffering at times from severe pain. In the evening he became drowsy and dull, and in the night he died after an attack of repeated convulsions.

It is unnecessary to dwell on the treatment, which was of no avail, for the most part, even in the way of palliation. Morphia injected beneath the skin in small quantities with extreme caution twice gave temporary relief. Once it failed utterly.

Autopsy (*abridged from Dr. Coupland's report*).—On removal of the calvaria, the vessels and sinuses of the dura mater were seen to contain fluid uncoagulated blood; the vessels of the pia mater were engorged, but there was no lymph on the surface of the brain; the inferior part of the temporo-sphenoidal lobe on the right side was superficially stained of a blackish-gray colour where the lobe had been in contact with the petrous portion of the temporal bone; the substance of the cerebrum was soft and vascular; the right lateral lobe of the cerebellum was the seat of an abscess the size of a walnut, approaching to the surface near the anterior and superior aspects of the lobe, in close contiguity to the pons Varolii. The contents of the abscess consisted of a creamy greenish pus of a highly fetid and gangrenous odour, the cavity being lined by a well-defined pyogenic membrane, and the surrounding nerve-substance dark-gray-coloured. No other abscess was met with elsewhere within the brain. All the cranial nerves were examined at their origin and appeared natural. The sixth and seventh nerves on the right side were particularly examined, with negative results. There was consider-

able adhesion of the dura mater to the cranium in the region of the right temporal bone, especially about the petrous part, and the separation of the membrane here revealed small collections of pus overlying extensive tracts of caries. The whole of the petrous bone was discoloured, presenting a blackish-gray tint, and at the upper part of its root of origin from the squamous and mastoid bones there was actual absence of osseous tissue, a thin-walled, pus-containing sac being here exposed. The right portio dura was carefully followed in its whole course from the internal auditory meatus throughout the aquæ ductus Fallopii; in its path over the superior wall of the tympanum it was bathed in pus, but the pus did not accompany it through the stylo-mastoid foramen. The chamber of the tympanum was filled with pus, and so were the cochlea and the entire labyrinth, which communicated freely with the abscess above described as existing at the root of the petrous bone. The membrana tympani was perforated, and the chain of ossicles, if present, were all hidden beneath the opaque semi-fluid contents of the tympanum. In the remaining organs nothing noteworthy was discovered beyond extreme engorgement of the lungs and kidneys.

The case above recorded, whether it deserves the name of Bell's paralysis or not, is at least an example of direct paralysis, owing to some lesion interfering with the competency of the facial nerve below the decussation, on its hither or peripheral aspect, the lesion and the paralysis being on the same side. Clearly the caries of the temporal bone is to be regarded as the primary fountain-head of all the ensuing mischief; whether it is to be looked upon as the main proximate source and determining cause of the facial paralysis is another question, and one which there are good grounds for answering in the negative. If the abscess in the cerebellum had power to paralyse the outward movements of the eyeball by implication of the sixth nerve, as unquestionably it had, may we not ascribe the immediate origin of the facial palsy in the main, if not exclusively, to the same abscess, involving in the same way the portio dura of the seventh nerve, where the two nerve-cords run side by side over the surface of the pons within the distance of a fraction of an inch from each other, or where in the region of their nuclei the juxtaposition is still closer? There are many points in the case that lead to this conclusion. In the first place, there is the singular slightness of the paralysis. The distortion of the features were never extreme; even the palsy of the orbicularis palpebrarum, though beyond the proportions presented in ordinary hemiplegia, fell far short of the degree of intensity displayed in average cases of Bell's paralysis, and of course immeasurably short of the consummation of that malady in lagophthalmia. Now, *primâ*

facie, there would be a fair presumption in favour of the idea that a nerve would be less liable to a damaging impression when softly cushioned on a bed of brain-substance than when cribbed and confined within a hard unyielding envelope of bone, and there encompassed by dense or disorganized material constricting its substance, or otherwise impairing its energies. Here, however, there was no such material within the bony channel; the nerve was simply bathed in pus, and appeared in this situation to have had fair scope for the discharge of its duties. All, or nearly all, the morbid influences would seem to have been concentrated upon it within the cavity of the cranium, and there to have determined, for the reasons above given, a modified or abortive form of Bell's paralysis. Secondly, the oscillations in the degree of the facial paralysis, the pauses, the remissions, and the exacerbations, so strangely contrasting with the steady advance of the disease as a whole, point strongly in the same direction. In particular, the symptoms of amelioration followed by those of relapse towards the close of the case would appear to be altogether inexplicable if they arose simply and solely from the condition of the temporal bone. They become at once intelligible when referred to changes in pressure or other collateral processes going on within the environment of the abscess. Lastly, there had been otorrhœa from childhood, and caries for an indefinite period prior to admission. Even the abscess was an old one, and the caries of necessity must have been older still, but the paralysis came on abruptly and concurrently with a violent outbreak of head-symptoms only five weeks before death. Now sudden and dangerous explosions after a long stage of smouldering are quite in character with the behaviour of abscess in the brain; they accord ill with the history of simple caries in the temporal bone, apart from abscess. The conclusion is clear: the paralysis must be taken along with the head-symptoms in the reckoning, and credited to the same proximate cause—the abscess; however strange it may seem that the diseased bone played no immediate part, or next to none, in the determination of the palsy.

CASE VIII

OTITIS, CEREBRAL ABSCESS, AND MALFORMATION OF THE HEART

Published in 'Medical Times and Gazette,' March 29th, 1873.

HENRY H—, æt. 4, a fresh-coloured child with a full face and a somewhat large head, was admitted February 23rd, 1872. Father healthy; grandfather died of consumption; mother delicate, supposed to have heart-disease—she was suffering from acute rheumatism at the time when the child was born; a brother, two years old, died with cerebral symptoms, in many respects resembling those observed in the present case. The child himself never had rheumatism nor any of the ordinary infantile diseases; he was a bright boy, intelligent beyond his years—indeed, his father used to make a constant companion of him in his walks; according to the description of his mother, he was always a "veiny child," but never presented the appearance of anything like cyanosis. Patient enjoyed good health until the age of two years, when he began to complain of pain on the right side of the head. The pain continued more or less unremittingly for some time, leaving the child, however, at last in apparent good health, although liable at intervals to recurrent attacks. Soon after the pain ceased in the first instance, or coincidentally with its cessation, the father noticed that a discharge came from the right ear, which he described as thick and yellow, with a horribly offensive odour. The discharge flowed continuously up to the date of the existing severe symptoms, which began eight days before admission, when the mother noticed the boy's left arm and leg trembling as he lay in bed; this trembling lasted for ten minutes, and when it ceased both limbs were found to be paralysed. A few hours afterwards he complained of pain in his head. The trembling returned and recurred with increasing frequency, the child grew restless and peevish, the pain in the head became worse from day to day, and he was brought into the hospital.

State on admission.—A loud, coarse (mainly, if not exclusively, systolic) murmur is heard at the heart's apex, beneath the axilla, and over the back of the chest. There is a purely systolic murmur over the whole of the upper thoracic regions in front, accompanied at the second and third left interspaces by a short diastolic bruit, inaudible to the right of the sternum. At the apex, and in the corresponding parts above described, the murmur is exceedingly long, and masks entirely the normal first sound. Sonorous râles very coarse and very abundant throughout the chest. Left arm and leg flaccid and powerless, but occasionally they manifest convulsive movements, and at times they are

quite stiff. The bowels are confined, and there has been vomiting of food for the last three days. Pulse 92, uneven in force and volume, and irregular in rhythm. A faint "cerebral streak" may be slowly elicited on the surface of the abdomen.

At 9.40 p.m. the child had a fit. First he gave a shrill scream, and then the limbs became rigid, and the breathing loud, harsh, and almost stertorous. He ground his teeth, and the affected arm and leg were sharply flexed, but there were no convulsive movements. Pulse 132, temperature 98°.

February 24th.—Left limbs perfectly powerless; muscles relaxed; cheeks intensely flushed. The child is exceedingly restless, tosses about from side to side, and puts his hand to his head, although he never complains of pain there. The inner surface of the auricle is slightly damp and exhales a fetid odour, but there is no free discharge from the meatus. Morning: pulse 72, small and irregular; temperature 98°. Evening: pulse 66, respirations 36, temperature 98°. At 9.30 p.m. the child became rigid and unconscious, but uttered no scream, and had no convulsions.

25th.—Passed a quiet night. Morning: pulse 72, respirations 24, temperature 98·4°. Evening: pulse 72, respirations 24, temperature 98°.

27th.—At 9.30 p.m. he again became unconscious, and died in the course of two hours.

Autopsy (thirty-eight hours after death, abridged from the report of Dr. Robert King).—On removing the calvaria and dura mater, there appeared at the centre of the right hemisphere, close to the longitudinal sinus, a patch of bluish discoloration, the size of a crown-piece, covered with a thin layer of creamy pus. A vertical section made through the middle of this discoloured patch laid open a large abscess, extending from the surface down to the lateral ventricle, and measuring fully an inch and a half in width. This abscess was evidently of old standing, enveloped in a strong cyst, and surrounded by indurated brain-tissue, in which the gray matter had wholly disappeared. Together with the lateral ventricle, the abscess was filled with yellowish creamy pus, which escaped from the infundibulum on the removal of the brain from the skull. There was no appearance of meningitis, nor of thrombosis in any of the venous sinuses of the dura mater; nor any evidence of pathological change in the structures of the ear, save a slight roughness of the floor of the right tympanum, a slight thickening of the mucous membrane of the right external auditory meatus, and a patch of necrosis in the upper part of the right mastoid process, about as large as a split pea. No connexion could be traced between the diseased bone and the abscess with its surroundings; the intervening structures were to all appearance healthy. The lungs were relatively small; they were œdematous, and the bronchial tubes contained puriform matter. The heart was relatively large, much of its increase being due to a greatly hypertrophied right auricle and ventricle. The right auricle was nearly as large again as the left, and the muscular substance of the right ventricle fell but little short of that on the opposite side. The foramen ovale was closed. No trace of a ductus arteriosus could be discovered. The pulmonary artery, for some distance above and below its bifurcation, was enormously dilated. On the other hand, the orifice of the vessel was greatly narrowed, puckered, and hardened by a fibro-atheromatous material—it would barely admit a No. 12 catheter, and was wholly destitute of anything like a true valvular apparatus. On opening the ventricles, they were found to communicate with each other by an aper-

ture in the ventricular septum, large enough to admit the tip of the little finger, situated at the extreme base of the heart, and formed by the absence of the uppermost portions of the septum, so that a probe could be readily passed from the right ventricle directly into the aorta. The mitral, aortic, and tricuspid valves all exhibited traces of atheromatous deposit, but as far as could be ascertained they were not incompetent. The aorta itself was narrowed at the top of the arch, where its section presented a diameter little more than half that of the pulmonary artery. The abdominal viscera were perfectly natural and healthy.

There was little or no difficulty in forming a rough estimate of this case. Malformation of the heart was in the highest degree probable; abscess of the brain was a certainty. Hemiplegia, when read by the light of the antecedent history, could only admit of this interpretation. The abscess had remained latent for a long period without giving rise to any pathognomonic symptoms. It might have been suspected, but it was not until eight days before admission that it declared itself unequivocally by breaking through the roof of the lateral ventricle, and inducing hemiplegia. Other circumstances, worthy of a passing notice, are the isolated position of the abscess away from the site of the original mischief within the bone, the slightness of the lesion in the bone itself, and the absolute integrity of the intervening brain-substance. Such circumstances are not unexampled in the history of encephalic abscess. The main difficulty centres in the exact interpretation of the auscultatory signs over the præcordia. I approach this question with extreme diffidence, for malformation is sorely bewildering to the auscultator; even at the post-mortem examination, with all the organs before him, he cannot always interpret the phenomena aright. At the apex, and over the whole of what I may call the mitral area, there was a loud, coarse, prolonged bruit, mainly, if not exclusively, of systolic rhythm. This bruit, I believe, originated in the aperture of communication between the two ventricles. The opposing masses of ventricular blood at the moment of the systole came into collision at this point with a rush, and gave origin to the murmur. It is impossible to speak of this murmur as significant of simple regurgitation or simple obstruction; it was the result of both together, or of processes allied to both, acting in both directions, towards the right and towards the left chamber—now one current predominating, and now the other. On the whole, then, the murmur became closely analogous to those which mark mitral and tricuspid insufficiency. Moreover, its seat of origin was not far distant from the auriculo-ventricular valves, and its area

of propagation precisely the same with the mitral area—before and behind. I have now to explain another murmur synchronous with the systole, loudest at the base, but heard extensively over the whole upper surface of the chest-walls in front. Clearly this was in the main the murmur of obstruction, due principally to the constricting membrane which replaced the undeveloped or disorganized valves of the pulmonary artery. Lastly, there was a faint and short, but perfectly distinct, diastolic murmur confined to the space between the second and fourth left costal cartilages, and wholly inaudible on the right side of the sternum, where we are sure to hear the murmur of aortic valvular incompetency. Clearly this was the murmur of pulmonic valvular incompetency, if valvular it can be called, where valve there was none, but only the rudiments and vestiges of a valve; at any rate, the structure was permanently open, and incapable of arresting the backward flow of the blood into the ventricle, just as a disabled valve might be. The murmur was short and faint because the column of blood was slender and the recoil of the artery feeble and inefficient. For the same reason the murmur is oftentimes altogether absent; indeed, however intelligible when present, it has been seldom if ever recorded among the physical signs of a variety of malformation in itself far from uncommon. The foregoing murmurs of pulmonic obstruction and regurgitation are, to the best of my belief, never heard, or only in the rarest examples, except in cases of congenital vice of development. The genesis of the malformation in our case is profoundly obscure. To assume any one of its constituent anomalies to be the first link in the chain, and from this to deduce all the rest in unbroken sequence on merely mechanical principles, would appear to be well-nigh hopeless. On the whole, perhaps it would be best and safest to regard two or more of the aforesaid lesions as coincident effects of one common cause inherent in the constitution of the mother or child, and in this respect the possibility of intra-uterine endocarditis with accompanying endoarteritis may fairly be taken into the reckoning. The mother, it must be borne in mind, was supposed to have heart-disease, and at the time of the child's birth was actually suffering from rheumatic fever. Now, although it may be urged that the foundation of all these changes must have been laid long before the few weeks preceding delivery, I hold this to be no fatal objection. The diathesis was there already in the constitution of the mother, and it is impossible to say when she first began to suffer from rheumatic

symptoms during the period of gestation. A very little rheumatism may give origin to endocarditis in a child, and it is easy to imagine that a still smaller amount in the maternal system may originate it in a fœtus. I have omitted all mention of the treatment adopted : the boy was doomed from the beginning, and the details of treatment would only be wearisome and uninstructive.

CASE IX

PNEUMONIA, DELIRIUM TREMENS, RUPTURE OF THE RECTI MUSCLES, ATROPHY OF ONE KIDNEY, AND HYPERTROPHY OF THE OTHER

Published in 'Medical Times and Gazette,' May 2nd, 1874.

C. B.—, æt. 40, a cellarman, was admitted March 12th, 1874. According to his own statement, his illness began six days before admission with a rigor and pain referred to the right front of the chest. In spite of the opportunities afforded by his occupation, he denies drunkenness altogether, and he has no relations to verify or disprove his own account of himself; but his whole aspect and all his gestures are unmistakably those of a hard drinker.

On admission, pulse 132, respirations 40, temperature 103·4°; chest imperfectly expanded; dulness in the right front; pupils small, nostrils quivering; tongue coated brown in the centre; face flushed; manner and movements nervous and agitated. In the evening the sputa were seen to be tenacious, frothy, and rust-coloured, and there was well-pronounced bronchial breathing heard over the right back from the spine to the angle of the scapula. Ordered a mixture containing carbonate of ammonia, acetate of ammonia, and tincture of squill, with a daily allowance of four ounces of brandy.

March 13th.—Passed a restless and delirious night, and wanders even now. Dulness in right front from apex to third rib; impaired resonance over the right scapula; marked tubular breathing throughout the corresponding area; faint sonorous respiration in right supra-spinous fossa; everywhere else the signs of bronchitis and œdema of the lungs. Urine scanty. Patient now acknowledges that he has had several attacks of delirium from drunkenness. Four ounces of port wine were allowed in addition to the brandy, and fifteen grains of hydrate of chloral administered every six hours. 3 p.m.: patient has just made a spring at the adjoining window; he charged straight at the perpendicular iron bar in front, and fell back with a crash on his bed. He was then removed to the delirium ward, where he became more composed and took his food, which he had before obstinately refused. Urine passed in small amount, smoky, and loaded with albumen. Two ounces of blood were withdrawn from the loins by cupping.

14th.—Spent a restless night. About two ounces of urine were collected separately from the motions, presenting under the microscope coarsely granular pigmented blood-casts and free blood-corpuscles in abundance. Chloral draught to be repeated every four hours, with the addition of ten minims of spirit of ether. Abdomen and loins to be fomented with infusion of digitalis of treble Pharmacopœia strength.

In the evening the pulse flagged; the expectoration disappeared; the patient had a fit of dyspnœa with lividity, and rattling was heard in the throat. Ordered a mixture, containing spirit of ammonia, spirit of ether, and syrup of tolu, every two hours.

15th.—2·30 p.m. the breathing became suddenly worse, and after a brief struggle ceased altogether.

Autopsy (abridged from the report of Dr Sidney Coupland).—On cutting into the abdominal wall the lower third of the sheath of each rectus muscle was found to be full of extravasated blood, partly coagulated, and each muscle was seen to be torn completely across at its point of narrowing about two inches above the tendon. At the seat of rupture the whole thickness of the muscle presented an appearance of extreme degeneration. It was exceedingly firm on section, and of a pale yellow, opaque, faintly granular aspect. The degeneration extended for some inches upwards, and was gradually replaced by healthy-looking fibres. Under the microscope the degraded fibres appeared to be much swollen, and wholly converted into irregular masses of highly refractive material, presenting, in fact, the characteristic features of the so-called waxy or vitreous degeneration of muscle in its most advanced stages. Other fibres, less advanced in degeneration, exhibited a granular aspect in place of the normal striation. None of the muscles of the thigh were affected. The muscular walls of the heart on both sides were thicker than natural; the valves normal. A few old adhesions existed at the apex of the right lung, and some recent readily detached adhesions over the whole of the upper lobe, which was covered by a thin false membrane. The apex was occupied by a mass of fibrous tissue forming a dense reticulation, while the rest of the lobe was thoroughly hepatized, presenting on section a pinkish-gray, finely granular surface. The middle lobe was also hepatized; the lower lobe compressed, non-crepitant, much congested, and tough, although floating in water. The upper lobe of the left lung was highly œdematous, the lower lobe much engorged throughout, and the whole organ non-crepitant. The right kidney was reduced to a congeries of cysts of the size of large grapes, the whole forming a mass no bigger than a racquet-ball. The cysts contained a clear amber-coloured fluid; they were lined by a dense opaque fibrous membrane, and communicated freely with one another. A small nodule of unaltered renal tissue existed at the upper part of the mass. The ureter was entirely obliterated, remaining only as a fibrous cord of the thickness of a goose-quill. The left kidney was notably hypertrophied, measuring five inches and a half by two and a half. Capsule non-adherent; surface smooth; substance unusually soft and flaccid; on section slightly paler than natural. Cortex in bulk apparently proportioned to medulla, but more opaque than normal. Microscopically, multitudes of blood-corpuscles were seen crowding the capillaries, and some of the tubes contained fibrinous exudation.

The foregoing case presents many features of interest and importance. It may be sufficient to select three points for a few words of commentary. First, the case illustrates well the concurrence of pneumonia and delirium tremens—an association so common that no physician of any experi-

ence, when dealing with a severe attack of delirium tremens, would ever omit to examine the chest for pneumonia. The relation would appear to be reciprocal; either of the factors in the combination may be the antecedent, and either may be the consequent. On the one hand, alcoholism, culminating in an outbreak of delirium, may develop pneumonia; on the other, a chance attack of pneumonia befalling an inveterate drunkard may upset altogether the unstable equilibrium of his nervous system, and give rise to delirium, just as an accidental injury or a surgical operation may. In the majority of instances apparently the delirium precedes and the pneumonia follows; in our case the order was reversed. Pneumonia, it is true, especially when it fastens upon the apex of the lung, may alone and unaided develop delirium, but that it was aided and abetted by drink in the present instance appeared to be incontestable in the judgment of all who witnessed the case. The second point is the hyperæmia of the kidney, the scanty secretion of urine, the hæmaturia, and the exudation within the uriniferous tubes. It is unnecessary to say how seriously the dangers of uræmia intensified the gravity of the prognosis and circumscribed the range of our remedies, especially in reference to the administration of opiates. The urinary disturbances are yet the more interesting, inasmuch as they all seem to have been the result of the malady itself, or the combination of maladies, and in no respect owing to any pre-existing disease in the kidney. Of course the atrophied organ is here put out of the reckoning altogether; it was reduced to an innocuous shell, and simply annulled—not diseased in the proper sense of the term. Neither could any enduring pathological change be discovered under the microscope in its hypertrophied fellow-organ. One fact, however, of some practical importance, the microscope did reveal. Although the kidney presented no signs of increased vascularity to the naked eye, the capillaries of the cortex were found to be overloaded with blood-corpuscles. The dwindling and sacculation of the right kidney must in all probability be ascribed to inflammation of the ureter consequent upon the passage of a calculus at some unknown date. The last point—the rupture of the recti muscles—is, perhaps, rather interesting than important. Clearly the exciting cause was the attempt to jump through the window, or the subsequent fall backwards; the predisposing cause, the waxy or vitreous degeneration of muscle described in the post-mortem records. A more picturesque name might be "glacier-like degeneration," the muscular

masses under the microscope in the advanced stages looking exactly like blocks or hummocks of ice huddled together one upon another unconformably. Finally, as there were no pathological appearances found in the small intestine, we may fairly exclude enteric fever, and number our own case among the many examples of so-called "myositis typhosa" unconnected with the fever that gave origin to the name.

CASE X

THROMBOSIS, WITH A NEEDLE FOUND IMBEDDED IN THE THROMBUS

Read before the Clinical Society of London, October 22nd, 1874. Published in their 'Transactions,' vol. viii.

Louisa T—, æt. 21, a housemaid, was admitted May 27th, 1874. She had suffered at times from globus hystericus and from palpitation, but never from rheumatic fever. Her last illness began two days before admission, with pains in the back and limbs, languor and loss of appetite, great thirst and constipation.

On admission, pulse 135, respirations 40, temperature 102·5°. Face extremely pale; pupils large; heart's action tumultuous; a systolic murmur at the apex; pulmonary sounds normal.

May 28th.—9 a.m. Pulse 128, respirations 32, temperature 103·8°. No sleep. Complains of pain and tenderness in the right iliac region and across the hypogastrium, but, indeed, the whole surface of the trunk appears to be unnaturally sensitive. Urine acid, of specific gravity 1023; contains about one-twentieth of its volume of albumen. 12.30 p.m. She is wildly delirious; the face is livid, and the lips have become dry and branny; the pulse amounts to 160, and the temperature to 105°. At 1.15 p.m. the temperature reached the maximum, 105·2°. At 2 p.m. she sweated profusely, the pulse marking 144 and the thermometer 104·5°. She was now calm and rational. At 10 p.m. the temperature, which in the afternoon had fallen to 100·2°, now rose again, and stood at 104·6°. The whole reading for the twenty-four hours gave a range of 5°, and presented three sharp-pointed peaks of elevation.

29th.—Slept five hours, with brief interruptions, and is now perfectly sensible and composed. At 10 a.m., after a severe rigor, the temperature reached 107·4°. From that point it sank spontaneously amidst drenching perspirations, until in the evening it fell to 100·4°. Ordered quinine five grains, and tincture of digitalis five minims every four hours, along with a daily allowance of brandy one ounce, and port wine four ounces. At 9.30 a second rigor, preceded by pain in the back, and followed by flushing and sweating; temperature 105·4°. At 11.30 p.m. a third rigor; temperature 105°. The whole range for the twenty-four hours amounted to 7·4°, and included three well-pronounced culminating points with one secondary projection.

30th.—At 10 a.m. an irregular sharply-defined crimson flush overspread the metacarpo-phalangeal joints of both hands. Brandy increased to six ounces daily. At 1.30 p.m. temperature 105°. Great hyperæsthesia, chiefly in the upper regions of the abdomen. At 6.15 temperature 104°, at 6.45 99·4°—a fall of 4·6° in half an hour. Whole range for twenty-four hours 6·6°, showing two pointed peaks and one pyramidal crest.

31st.—Slight delirium. Complexion pale and earthy, but without a trace of jaundice in the conjunctiva. Tongue dry and brown. Sonoro-sibilant râles throughout the chest. Brandy increased to ten ounces daily, and quinine to ten grains every four hours, or one drachm in the course of the day. At 1 p.m. face coloured purple in patches like the mottling on the skin of a corpse. At 9 p.m. a rigor; at 10 p.m. deafness and noises in the head; temperature 105°, the maximum for the twenty-four hours. Range 5·2°, comprising three well-marked apices of elevation.

It is simply impossible to give the details of the residue, diffused, as they are, over a space of fifty-three days. In the main the disease advanced at a singularly slow and monotonous pace, each succeeding day for the most part, but not invariably, repeating its predecessor. The most frequent and the longest breaks in this monotony took place in the month of June, when the girl really seemed to have some enjoyment in life. On nineteen days in the series of fifty-three there occurred distinct and definite rigors; on four days mere chilliness; on seven vomiting. Sweating as a rule followed shivering, and sometimes appeared to replace the rigors altogether. In the same way vomiting or faintness either concurred with the paroxysm of shivering or played the part of its representative. Whatever the mode of manifestation, the temperature at the time of the outbreak almost always culminated in a peak; on six days at 105° or more, on twenty-three days at 104° or more. On the other hand, the remissions kept steadily above the normal standard, save on two occasions only, until June 23rd. From that date, during the month preceding death, they were found to be subnormal, or even below that level, on seventeen days. One of these days presented a maximum of 105·6°, and a minimum of 96·6°, yielding a range of 9°. The lowest reading recorded is 95·6°. During the last week of life the exacerbations subsided, contracting the range within a narrower compass, and giving more the character of continuity to the mean average, whether this result be regarded as owing to the near approach of death or to the wear and tear of exhausting diarrhœa at the close. As to the course of events in general, one paramount circumstance requires to be noted in the foremost place. Phlegmasia alba, with conspicuous enlargement of the superficial veins, appeared in the left lower extremity on June 8th, the fifteenth day of the disease, and in the right on July 8th, the forty-fifth day. In both extremities it remained to the end. A rather noteworthy circumstance is the presence of signs and symptoms, closely resembling those of pericarditis, from June 8th to 16th. A third is the presence of pain in various regions of the spine, usually of an aching character, causing faintness or actual syncope, and sometimes, but not always, coinciding with rigors or with a high degree of pyrexia. For the rest suffice it to say, that after severe suffering from diarrhœa, with pain and tenderness in the epigastrium and abdomen at large, and after a prolonged period of delirium or unconsciousness, now and then broken by a few lucid intervals, death took place on July 23rd, fifty-nine days from the date of invasion. As for the treatment, the dose of the quinine was raised on June 21st to fifteen grains every four hours, or to a drachm and a half in the day, while stimulants were liberally administered throughout, and anodynes, hypnotics, and astringents from time to time, to meet the requirements of the case.

The blood was twice examined under the microscope during life. The

white corpuscles were found to be inordinately numerous, and the red shrivelled and otherwise deformed, but no bacteria or microzymes of any kind could be distinguished. The clots discovered after death were not examined.

Autopsy (from the report of Mr R. H. Lucas).—In the head were found a number of minute extravasations beneath the lining membrane of the third ventricle. No softening or abscess could be detected anywhere; the membranes were not congested, nor was there any lymph upon them.

In the thorax there was sub-pleural ecchymoses observed all over both lungs, but not over the chest-walls. The substance of the lungs was œdematous and pale for the most part, but congested at the bases. There was no abscess or appearance of consolidation to be discovered. The bronchial tubes were not inflamed. The cavity of the pericardium contained about one ounce of clear straw-coloured fluid, and there were one or two white patches over the left ventricle firmly adherent and tough. The left auriculo-ventricular orifice measured four inches. The mitral valves were somewhat thickened, and presented a chain of small vegetations along the auricular margins. The substance of the heart was flabby. *In the abdomen* the vena cava inferior, from the junction of the renal veins to its bifurcation, was filled with a partially decolorized blood-clot adherent to the wall of the vessel, but easily detached from it. A channel containing a recent coagulum extended down the centre of the plug. At the bifurcation the vessel was filled with dirty-brown matter, and in the midst of this a needle was noticed resting in an oblique direction with its eye pointing downwards and slightly to the left, in close relation to an opening in the back of the vein about an eighth of an inch in diameter. Immediately above this another opening existed measuring one quarter of an inch. Through these apertures the body of the fourth vertebra could be felt denuded of its periosteum. The left common iliac vein was very large and filled with a brownish-red clot of moderately firm consistence and slightly adherent. The left external iliac and left femoral were each as small as a crow-quill, and each completely occluded by a whitish fibrous plug, which could not be detached from the wall of the vessel. The right common iliac, right external iliac, and right femoral, were obstructed and narrowed by a tough, fibrous-looking, firmly attached layer, its inner surface covered with thick patches of coarse yellow material which could be easily stripped off. The vessels, however, were not completely occluded. Situated immediately to the left of the third lumbar vertebra, and the intervertebral substance below it, was an abscess, the walls of which extended to the margin of the psoas muscle, and involved the outer coat of the left common iliac artery. On opening this abscess, which contained about half an ounce of pus, a second needle was seen lying obliquely across the body of the third lumbar vertebra on the left side, with its point directed downwards and to the right. The substance of the vertebra, however, was not eroded. The vessels in this situation were all matted together, and were with difficulty separated. Liver engorged with blood, especially in its left lobe. The spleen, large and firm, weighing ten ounces, presented a yellow-coloured infarction about the size of a marble, raised above the surface and softening in the centre. A few smaller masses of unsoftened yellow deposit were found scattered here and there about the circumference of the organ. Intestines much injected. Mucous membrane the seat of extensive catarrh. Kidneys normal.

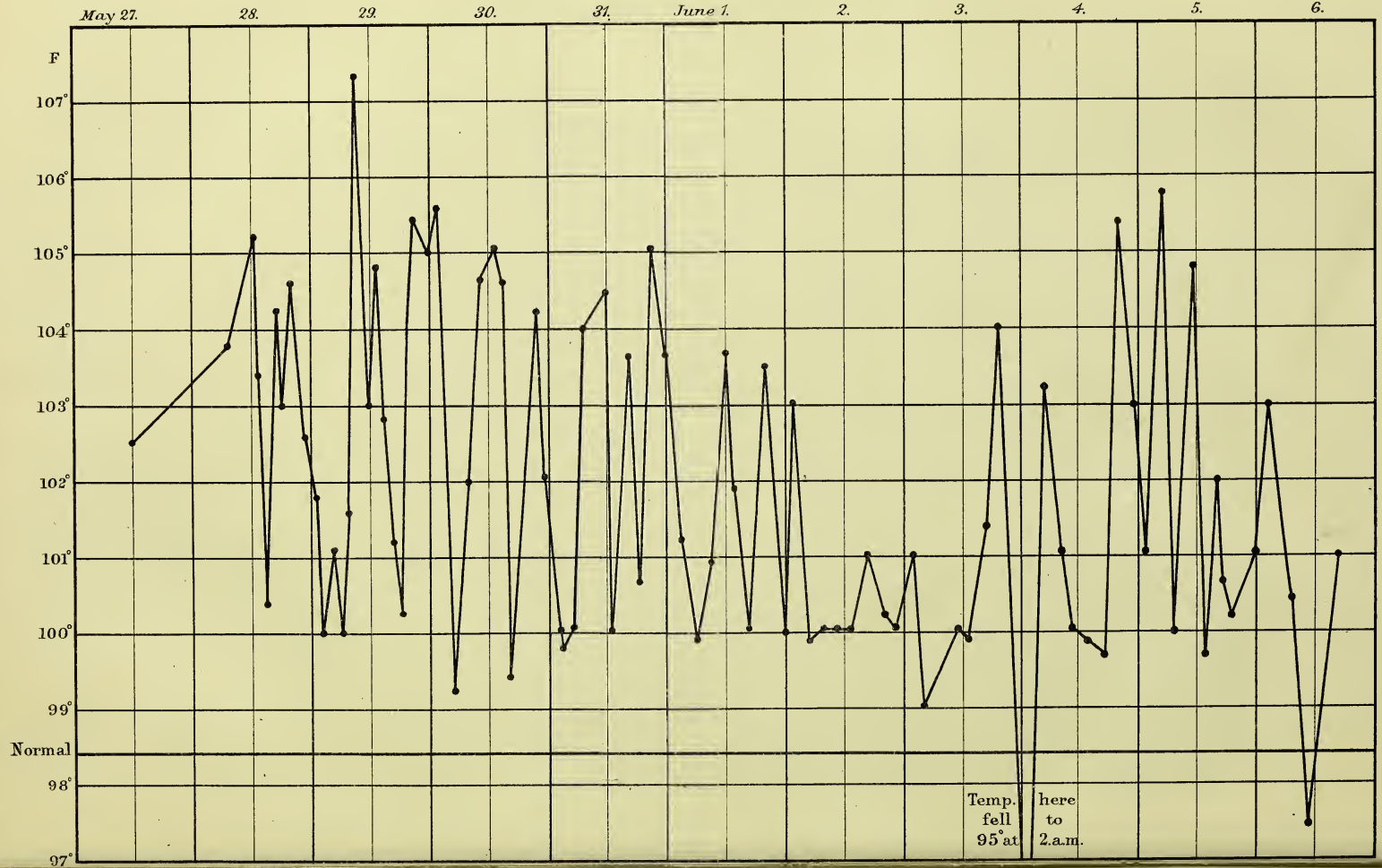
The phenomena presented by the foregoing case, during life and after death, are alone sufficient to entitle it to a place in the records of disease, whatever opinions may be formed with reference to its pathology, and by whatever name it may be designated. Many would call it a case of pyæmia; or, if not pyæmia, at all events septicæmia: some, perhaps, would be content to class it under the head of simple thrombosis, with accompanying abscess in the environment of the vessels involved. It is certainly remarkable that, in a case so strikingly characterized by the very conditions presumed to give origin to pyæmia proper, so long protracted, and so faithfully representing pyæmia in its general features, no unequivocal secondary deposits could be discovered anywhere. It is true there were spots of sub-membranous hæmorrhage found after death, but these are not secondary deposits as commonly understood, nor are they peculiar to pyæmia in the limited sense of the term. Again, it is true the spleen was large and contained infarctions—one of considerable size and prominence, surrounded by the usual halo of blood and already in course of softening; but even these appearances are at best indecisive in the presence of vegetations on the valvular structures of the heart. It cannot be said, therefore, with absolute assurance of certainty, that there were any deposits at all secondary to pyæmia, and it may well be to this very immunity of the viscera and vital organs that we must ascribe the long duration of the disease. May we ascribe the immunity itself in any degree to the influence of quinine in controlling the processes of infection, inflammation, and suppuration, as an antiseptic and antiphlogistic? It would really appear at first sight as though there were some truth in this hypothesis; but in order to lay down the law on the question at large, we should require a hundred cases, fifty treated with quinine, and fifty without. Whether the quinine made any strong impression upon the temperature is a point far easier to raise than to resolve. During the exhibition of the remedy in daily aggregates of a drachm and a drachm and a half, the mean average undeniably ruled lower; but there were times when the fever-heat culminated at the same high level as before—if we exclude the solitary rise to 107·4°—and there were times when the oscillations and aberrations were as frequent and fitful as ever. The low scale of the last eight or ten days, and in particular the depth of the remissions, cannot with certainty be placed to the credit of the quinine; indeed, throughout the whole career of the case it is impossible to say

how many of the phenomena were the results of medical interference, how many mere natural stages in the march of the malady. I have ignored digitalis altogether; the dose was not large enough to be of any material moment. Perhaps some persons may make the same remark on the quinine itself. Mere reduction of temperature, however, is here a matter of comparative insignificance. If it be regarded as part and parcel of the antiseptic and antiphlogistic operations of quinine, then it is chiefly valuable as an index and measure of good accomplished. If it is to be looked upon independently in the light of an agent for the accomplishment of good, it must surely be well-nigh powerless in the face of so fearful a plague as pyæmia or septicæmia, engrafted as they are on lesions and morbid conditions far beyond the reach of mere refrigeration. To give quinine with this purpose would only amount to lopping off a branch, when our endeavour should be to strike home at the root.

As for the presumed origin of all the mischief, the swallowing of the needles, it must have been owing to inadvertence and momentary distraction of mind. Hysterical the girl undoubtedly was in some slight degree; but amid all the mad freaks of incorrigible hysteria it can hardly be imagined that a woman would deliberately swallow needles, although women are well known to have stuck pins and needles in various parts of the body. The girl was in the habit of holding these things in her mouth, and in a moment of forgetfulness, under the excitement of any passing circumstance, she might easily swallow one. In truth, her family yet preserve a shawl-pin which she swallowed in this way, on being hastily called by her master. For thus much—for the element of excitability and absence of mind—the hysteria may fairly be held responsible, but for no more.

As for the path pursued by the needles, it is indeed superfluous to speculate on anything so inconstant as the behaviour of sharp metallic substances when once they make their way into the body. Wherever the point of entrance may be, they may lodge almost anywhere, or they may emerge almost anywhere. In our case there was a simple and straighforward course for the needles to take. Most probably they penetrated the œsophagus and followed the track of the spinal column until they arrived at the bifurcation of the cava.

CHART OF TEMPERATURE. — Louisa T.



CASE XI

CIRRHOSIS AND CARCINOMA OF THE LIVER, WITH HEART DISEASE AND ASCITES

Published in the 'Medical Times and Gazette,' May 23rd, 1874.

Henry A—, æt. 60, a labourer, was admitted into the Middlesex Hospital on December 9th, 1872. There was no history of cancer in his family. He had led an intemperate and somewhat dissolute life in his youth, and had contracted syphilis. At twenty years of age he had an attack of acute rheumatism. During the past fourteen years he had suffered from winter-coughs, with breathlessness on exertion, and occasional palpitation of the heart. He had been habitually a martyr to indigestion, with flatulency, foul tongue, and want of appetite. About thirteen months before the date of admission he was seized with sharp pain in the right hypochondrium, and was ill for a week. About five weeks before the same date he first observed swelling of the abdomen and lower extremities.

State on admission.—Patient pale, and much emaciated. Enormous distension of the abdomen, which gives free and extensive resonance over the uppermost part, but is decidedly dull in the flanks, where fluctuation may be readily felt. The wave may also be transmitted, but with some difficulty, across the breadth of the abdomen. Considerable œdema of the lower limbs, where the veins are varicose. Heart-sounds both unnaturally rough at base and along great vessels; first sound alone roughened at apex; same deficient in clearness at back. Over the third right cartilage the roughness of the diastolic sound appears at times to end in a short murmur, following the sound and propagated in the direction of the apex. Præcordial dulness abnormally extensive; slight cough and dyspnœa; pulse sharp and thrilling.

December 10th.—Girth at umbilicus forty inches. No albuminuria. In the evening the breath was short and the pulse irregular.

16th.—Orthopnœa during the night; abdomen immensely distended, veins enlarged, and surface glistening. The urine showed a trace of albumen. Paracentesis was now performed, and six pints sixteen ounces of clear straw-coloured fluid were withdrawn. After operation, girth thirty-nine inches; pulse 108; temperature 99°.

17th.—Fluid has been oozing from the wound throughout the night. At 9 p.m. continued oozing; intense thirst; pulse 120; temperature 101°; nausea, but no vomiting.

18th.—Continued oozing; temperature 99°. In the evening the fluid ceased to escape. Temperature 98·8°; appetite improving.

19th.—Girth forty inches. Liver surface, rough and nodular, can be felt over the space of two or three inches below the margin of the ribs.

22nd.—Girth forty inches and a half; temperature 99°; pulse 96. Slight tenderness of abdomen; free fluctuation everywhere; veins again beginning to be conspicuous.

23rd.—Pulse 102, temperature 100°. Fluid has been filtering from the wound since 2 a.m.

24th.—Continued escape of serum throughout night. Girth thirty-nine inches; pulse 116. There are now both pain and tenderness in abdomen, and in the evening he vomited a quantity of greenish matter.

25th.—Slept fairly; pain relieved; no oozing; girth thirty-eight inches and a half; pulse 130. Pain and vomiting returned in the evening.

26th.—Pain continues; it is much increased by pressure. Vomiting also continues; no escape of fluid; girth thirty-nine inches and a half.

27th.—Passed a restless night; pulse 130.

28th.—Another restless night; pulse 126; girth forty inches; intense thirst. Legs drawn upwards, features pinched, nostrils quivering, cheeks projected with every expiratory act. At 12.15 p.m. he suddenly sank, and died in a few seconds.

Autopsy (abridged from Dr Sidney Coupland's report).—All the heart's cavities were full of blood, especially the right, which were much dilated. Pulmonary and tricuspid valves healthy. The segments of the mitral valve were slightly thickened. In the aorta there were only two segments of the semilunar valves, the free margin of each measuring one inch and a half. Their upper surfaces were covered with firm calcified growths, but the valve itself was competent to the water-test. Right lung emphysematous anteriorly, posteriorly gorged with blood. Left lung also gorged and œdematous. The abdominal cavity contained six pints six ounces of turbid straw-coloured fluid. The pelvic peritoneum in parts, and some of the coils of the small intestines, were injected, but there was no lymph attached to the serous surface, and there were no adhesions anywhere. The liver was of great size, owing to immense enlargement of the right lobe, which on section presented hardly a trace of true gland-substance remaining; the whole lobe being constituted of white and yellowish-white material, forming irregular tracts of various size, the larger masses being soft and brain-like in colour and consistence, while the smaller were firm and overgrown with fibrous tissue. The left lobe, coarsely hobnailed on the surface, was apparently free from cancerous infiltration; the isolated bile-stained lobules representing the only normal gland-elements left. The microscopic appearances were those of carcinoma and cirrhosis, separate or conjoined. Spleen large and fibrous; capsule pale and thickened. Kidneys mottled and streaked on section; surface smooth.

The case recorded is interesting pathologically as an example of carcinoma rapidly developed and engrafted as an overgrowth on long-standing cirrhosis. Again, the physical signs in the chest were peculiar, and in this connexion the state of the heart is interesting in a clinical point of view. I was much struck during life with the characters of the pulse,

which pointed strongly to the presence of aortic valvular incompetency, and with the results of auscultation, which seemed to confirm that suspicion, but at times only, and not invariably. Now and then, over and above the uniform asperity of the second sound, there appeared to be a sequel or appendage thereto—a prolongation of the sound with all the characters of a short but genuine murmur. All this is exceedingly well explained by the post-mortem appearances. There were only two segments to the semilunar valve of the aorta, and these were thickened and covered superiorly with calcified masses; the valve, however, seemed to be competent when tried by the water-test, whatever the value of that may be. In my opinion the valve during life was at times competent, at times incompetent; hence, one day we found simply a roughness in the diastolic sound, due to calcareous growths; on another we traced the prolongation aforesaid, due to an intermitting reflux of blood from artery to ventricle. A valve may be incompetent in varying degrees under a variety of circumstances connected with the ever-changing amount of blood it is called upon to hold in check from time to time; nay, if not seriously damaged, it may be competent at one time and incompetent at another. This intermittent incompetency is acknowledged to be true of the auriculo-ventricular valves; it is true, also, I am convinced, in disease of the aortic valve.

Finally, the case has a practical bearing. Is there any advantage in the oozing of fluid which sometimes follows paracentesis abdominis? I have seen great advantages many times. One case of ascites under my care made a marvellous recovery, owing, as it appeared, in great measure to continued outflow of serum from the wound in the abdomen. Is there any disadvantage or danger in prolonged percolation of fluid? I know of none, as a matter of experience; unless, indeed, the present case be an example in point. Unquestionably the leakage staved off the necessity of a second tapping, but from the accompanying evidences of irritation, and even inflammation, there are grounds for surmising that it may have been in part responsible for the peritonitis, whereof we discovered incipient manifestations after death. The oozing had ceased in the first instance on the 18th, and its cessation had been succeeded by a decided improvement. It recommenced on the 23rd. All then depends on the date of the first symptoms that betrayed the presence of peritonitis. Now, it is impossible to say whether they began on the 22nd or on the 24th, and

therefore our own case is only an equivocal example. As for the remaining cases that have occurred under my care, perhaps they are insufficient in number to determine so important a point in practice. If, on a larger comparison of accumulated examples, the process of oozing should prove to be advantageous, it is a fair question to consider how far it can be artificially produced and maintained, of course without incurring the danger of admitting air into the peritoneal cavity, or of inducing peritonitis by propagation of morbid action from the wound. This, however, is a problem which must be left for the surgery of the future to solve.*

* The problem has been solved. Dr. Southey's fine drainage-tube would seem to meet all the requirements of the case.

CASE XII

INCOMPETENCY OF THE AORTIC AND TRICUSPID VALVES. DEATH FROM PERFORATION OF THE DUODENUM

Published in 'Medical Times and Gazette,' February 8th, 1873.

Henry G—, æt. 62, originally admitted under Dr Cayley, in October, 1872, was readmitted, under my care, on December 3rd in the same year.

History.—Father and mother both dead; cause of death unknown. Patient belonged to a family of seven; one sister alive and healthy; all the rest are said to have died of gout or rheumatism. He himself had suffered from syphilis and frequent attacks of gonorrhœa, and for some years past from stricture of the urethra and incontinence of urine. He had led a most intemperate life. Never had rheumatic fever. For the last few years he had been much harassed by a cough, aggravated during the winter months, and by breathlessness, especially on exertion. For four years he had been an out-patient of this hospital. When admitted under Dr Cayley he presented the signs and symptoms of bronchitis, emphysema of the anterior portion of the lungs, condensation of the posterior regions, more particularly on the left side, enlargement of the heart, and incompetency of the aortic valves. After remaining under Dr Cayley's charge for a short time the condition of the lungs rapidly improved, and he left the hospital on November 11th, 1872. On readmission the following particulars are recorded:

December 3rd.—Patient looks paler and thinner, and his lips more livid than during his previous residence. There is a double murmur everywhere over the præcordia and great vessels, loudest to the left of the sternum at the third cartilage, but very distinctly heard at the apex. No unequivocal murmur at the back of the chest. Præcordial dulness reaches the third cartilage above, below it extends two inches beneath the nipple, laterally one inch or more beyond the mammary line on the left side, and rightwards nearly an inch beyond the sternum. There is considerable œdema of the lower limbs. Pulse 84, jarring and collapsing; temperature 99·4°.

4th.—Slept well. Slight cough. Bowels confined last six days. Urine of specific gravity 1025, non-albuminous. External jugulars large and beaded, especially the right vessel, which pulsates vigorously, the pulsation almost disappearing when pressure is made just above the clavicle.

5th.—Passed a noisy and restless night, calling clamorously for wine and brandy. Sonoro-sibilant râles everywhere. Harsh, high-toned, tubular breathing over the left scapula and below. Faintly bronchial breathing, with slight dulness at the right posterior base. Lips parched

and deeply livid; tongue dry, red, and horny. Pulse small and soft; much of its characteristic jar is gone. The bowels have not been open for a week. Died at 11 p.m., rather suddenly.

Post mortem (*as reported by Dr Robert King*).—Both lungs much congested, and their bases, especially the left, nearly solidified by patches of apoplexy and lobular pneumonia. The anterior margins and apices of the lungs were highly emphysematous. The heart was of large size, and showed white patches on its surface. The right cavities were much dilated, the tricuspid orifice measuring fully five inches in circumference. Each ventricle contained a small quantity of black clot. The aorta was a mass of atheroma, and was much dilated; the aortic valves being greatly thickened and quite incompetent. The abdominal cavity contained a large quantity of dirty fluid and fæcal matter, which had evidently escaped from an oval opening with blackened, thickened, and rounded edges, situated in the duodenum, close to the pylorus, and large enough to admit the tip of the little finger. The whole of the intestines were red and injected, and some of the coils adherent. On slitting open the bowel three additional smaller ulcers were found in the duodenum, close to the one in which perforation had occurred. The liver was softer than natural, but evidently in places cirrhotic. The gall-bladder and ducts appeared to be healthy, and free from gall-stones. The kidneys were rather large, pale, and soft; their capsules were non-adherent. The spleen was very small and soft. The urinary bladder was much thickened and sacculated; and in the urethra was a stricture barely admitting an ordinary probe, and situated just anteriorly to the bulbous portion.

The foregoing case is presented as an example of what may be called the surprises of medical experience. When first encountered it appeared indeed to be grave, and not at all unlikely to come to a close during the ensuing winter, but no one entertained the remotest idea of its closing in the manner revealed by the post-mortem examination. To the last moment the man was imagined to be suffering from heart-disease, with its accompaniments, ordinary or extraordinary, and from nothing more. Not a single sign or symptom pointed expressly in the direction of the abdominal cavity or the alimentary canal, save only constipation of a week's date; and that surely could not be regarded as significant of ulcer in the duodenum; still less could it be held responsible in any way for the fatal issue. There was no complaint of sudden pain or tenderness in the abdomen; no sensation of anything giving way within; no instantaneous collapse; only a progressive sinking, *as it seemed*, under the associated influences of apnœa and asthenia, with corresponding morbid changes in the lung-tissue, in the heart's cavities, and in the pulmonary arteries. And yet the main cause of death was perforation of the duodenum! On the whole, the best way of explaining the latency of the

peritonitis would appear to be the following :—The man was so far gone already, and so utterly prostrated by the pre-existing heart and lung disease, that the symptoms of perforation were lost in those of exhaustion, and failed to express themselves in their proper characters. The nervous system is clearly the prime paralysing agent in the causation of collapse, and when that system is itself paralysed by the long wear and tear of one disease, it may easily become dead to the strongest impressions of another. Nevertheless, although the nature of the final malady was undetermined and unsuspected during life, we may now, I think, determine with a fair amount of probability the date of the perforation. In all likelihood that event occurred on the noisy night—that of December 4th and 5th. Of course vociferation and noise apart from delirium have no special significance in disease, but at least they were signals of distress, and in this point of view may be looked upon here as the first outward and perceptible tokens of alarming mischief within. The appearances of the tongue and the failure of the pulse may now, as we read the case backwards, be ascribed to the same cause, but they could hardly have been so interpreted beforehand. The date of death accords extremely well with the assumed date of the perforation; death ensued near midnight on the following day, after an interval of about twenty-four hours. As for the origin of the ulcers, there were no vestiges of a burn and no evidences of carcinoma anywhere.

London, New Burlington Street.
November, 1879.

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